EVALUATION OF CASES WITH CARCINOMA STOMACH

Dissertation submitted to the

TAMIL NADU DR.MGR MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

for the degree of

MASTER OF SURGERY

IN

GENERAL SURGERY

UNDER THE GUIDANCE OF

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TIRUNELVELI MEDICAL COLLEGE
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2013

BONAFIDE CERTIFICATE

This is to certify that this dissertation titled **"EVALUATION OF CASES WITH CARCINOMA STOMACH"** is a bonafide record work done by **Dr. SUMATHI. M** under my direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MS (General Surgery).

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INTRODUCTION

Gastric Carcinoma, one of the oldest disease which dates back to 980-1037 BC. Aricena was the great person who gave first inputs regarding Gastric carcinoma. In 1761 Morgagni, was the person, who provided elaborate findings related to carcinoma stomach. Passing through the years, it has become, one the leading cause which contributes to death across the world.

The various reviews across the world now states that gastric malignancy is on declining pattern but, in India malignancy of stomach still a major factor which has an impact over low income and people who are below poverty line.So this study was chosen to find out the various factors which will affect the occurrence of Gastric cancer

Malignancies related stomach has maximum occurrence among southeast Asian countries. Among these, Japan bears the major brunt. This may be mainly due their food habits, radiation, obesity. In India, Gastric carcinoma is one the leading disease which causes morbidity and mortality. North-eastern states, especially Mizoram is the leading contender in occurrence of carcinoma stomach.

The main contributing factors for the increased incidence of gastric cancer will be food habits and environmental factors. Among food habits,

having salt content more in food and, increased consumption of grilled foods are the major determinants. In the environmental factors smoking, alcohol consumption, tobacco chewing are the main culprits for the occurrence of gastric malignancy

The etiopathogenesis of gastric malignancy will be mainly related to interaction between genetic and environmental factors. Diet and H.pylori will be the major contributing factors with regarding to environmental causes. With recent advancements in molecular biology enlights us, the concealed facts regarding genomic association of gastric malignancy. Over expression of abnormal genes and .lack of heterogenisity in tumour suppressor genes are the important factors with genetic association

Among the types of gastric malignancy adenocarcinomas predominates. The rest of the types will be lymphoma and stromal tumours. Adenocarcinoma has two histological types. Well differentiated or diffuse type and intestinal or undifferentiated types.

The morbidity and mortality related to gastric malignancies is mainly related to the time when the patient comes to the hospital. Early detection and intervention will increase the five year survival rate.

AIMS AND OBJECTIVES OF THE STUDY

1. To study the prevalence of carcinoma stomach as occurring in Tirunelveli Medical College Hospital.
2. To find out the etiology and risk factors for carcinoma stomach
3 To study the presenting clinical signs and symptoms of carcinoma stomach
4. To study the anatomical location of carcinoma stomach with respect to the age and sex
5. To analysize the histopathological type in relation to the site growth and age of the patient
6. To study the surgical modalties of treatment.

REVIEW OF LITERATURE

Gastric malignancy is one of the leading cause of cancer related deaths. Its prognosis tends to be poor with cure rates little better than 5–10%, although better results are obtained in Japan where the disease is common. Gastric cancer is actually an eminently curable disease provided that it is detected at an appropriate stage and treated adequately. It rarely disseminates widely before it has involved the lymph nodes and, therefore, there is an opportunity to cure the disease prior to dissemination. Early diagnosis is therefore the key to success. Unfortunately, the late presentation of many cases is the cause of the poor overall survival figures. The only treatment modality able to cure the disease is resectional surgery. The occurrence of gastric cancer differs across the world. Various countries showed different datas with relation to its incidence. Of these Japan is the forerunner in terms of cases per one lakh population. It closely comes around 70 cases/population/year .Other western countries has lesser incidence rates when compared to japan.Eastern European countries and China closely matches japan in terms of occurrence of gastric cancer.

In India, the occurrence of gastric malignancy varies region wise. According to the National Cancer Register, North eastern states has the maximum number of population with gastric cancer. It has surpassed

Kashmir,in terms of incidence. Among north ,south difference ,south Indian population are more vulnerable to have gastric cancer. This may be due to diet related factors, which play a major role.

The occurrence of gastric cancer differs across the world. In general, men are more affected by the disease than women and, as with most solid organ malignancies, the incidence increases with age. At present, marked changes are being observed in the developed countries in terms of the occurrence and sites of gastric malignancy and the population affected, changes that to date have not been observed in Japan. First, the incidence of gastric cancer is continuing to decrease, at a rate of about one percentage per year, with the reduction exclusively affect in carcinomas arising in the body of the stomach and the distal stomach. In contrast, the incidence of carcinoma in the proximal stomach, particularly the oesophagogastric junction, appears to be increasing .Malignancy occurring in the distal and body of the stomach is most common in low income groups, whereas the increase in proximal gastric cancer seems to affect principally the people belonging to the higher income group . Proximal gastric cancer does not seem to be associated with H. pylori infection, in contrast with cancer affecting the body and the distal stomach.

HISTORY

1875 Sidney Jones in London publishes the first successful gastrostomy for feeding.

1879 Paen performed distal gastrectomy and gastroduodenostomy. The patient died 5 days later.

1880 Rydygier ressected a distal gastric cancer, and the patient died 12 h later.

1880 Billroth ressects distal gastric cancer and performs gastroduodenostomy (Billroth I).

1881 Anton Wolfler performs loop gastrojejunostomy to palliate an obstructing distal gastric cancer.

1884 Rydygier reports an unsuccessful gastrojejunostomy for benign gastric outlet obstruction.

1885 Billroth performs a successful distal gastrectomy and gastrojejunostomy (Billroth II) for gastric cancer

1980–2000 Japanese surgeons and other surgical groups from East Asia demonstrate that more aggressive lymphadenectomy may improve survival in patients with gastric cancer.

1990–current Evolving role of laparoscopic techniques in the treatment of surgical gastric disease

ANATOMY OF STOMACH

Development: The caudal part of foregut shows a fusiform dilation with anterior and posterior borders,along with right/left surfaces, This partiscalled stomach.It rotates 90 degree clockwise,so that left surface faces anteriorly.the posterior border of stomach grows faster ,forming the greater curvature.

The stomach rotates anteroposterior axis ,so that distal or pyloric part moves to right and proximal or cardiac part moves to left side.

The 90 degree rotation of stomach along the vertical axis pulls the dorsalmesogastrium to the left and forming the lesser sac or omental bursa.

The embryologist describes the stomach as a fusiform dilatation of the foregut, beginning at the gastroesophageal junction and ending at the gastro duodenal junction.

The anatomist describes the stomach in terms of its several parts, such as the gastroesophageal junction, cardia, fundus, body, antrum, pyloric canal, and sphincter, but also accepts the stomach as a distinct entity, a well-defined organ that is easily visualized, dissected, and demonstrated.

The physiologist and gastroenterologist describe the endocrine and exocrine stomach, treating the organ as two units, proximal and distal:

Proximal stomach - The proximal stomach (fundus and body) receives and temporarily stores gastric contents. It is the home of the parietal cells which secrete acid and intrinsic factors, as well as the home of the cells that produce Group 1 pepsinogen.

Distal stomach - The distal stomach (antrum and pylorus) mixes and propels the gastric contents. It also contains the area of the pyloric glands which produce the hormones gastrin and somatostatin.

The distal stomach and pylorus are likely to reveal vital inhibitory mechanism in the regulation of gastrointestinal movement; .

The pathologist recognizes three subdivisions of the stomach: the fundus, body, and antrum. These provide the basis for descriptions of localized gross pathology.

The radiologist, in practical terms, locates the gastroesophageal junction just "below the junction," and refers to the first part of the duodenum as the duodenal bulb.

With reference to surgery, stomach was divided into proximal gastric unit and distal gastric unit. The proximal unit comprises of upper stomach, distal oesophagus and diaphragmatic hiatus.

The distal unit includes antrum, pylorus , along with the first part of the duodenum.

PROXIMAL GASTRIC SURGICAL UNIT

The proximal gastric unit consists of the distal esophagus, esophageal hiatus, and proximal stomach. The esophagus joins the stomach in the abdomen, just below the diaphragm. The length of the abdominal esophagus is from 0.5 to 2.5 cm

The abdominal esophagus lies at the level of the 11th or 12th thoracic vertebra, perhaps lower in tall, asthenic subjects, and higher in short subjects.[31] Its relationships to surrounding structures are:

Anterior: Posterior surface of left lobe of liver

Posterior: Right crus of diaphragm, aorta

Right: Caudate (spigelian) lobe of liver

Left: Fundus of stomach

Relations of the Proximal Gastric Surgical Unit

The proximal gastric surgical unit has relationships with the lesser and greater curvatures, the upper part of the lesser sac (omental bursa), and the gastroesophageal (G-E) junction.

The lesser curvature of the body, the G-E junction, and the abdominal esophagus are attached to the hepatogastric ligament and its contents.

The greater curvature attaches to the upper part of the greater omentum and the several related splenic ligaments. The anatomic entities of the G-E junction will be found in the chapter on the esophagus.

DISTAL GASTRIC SURGICAL UNIT

Gastric Antrum

In the opened stomach, the antrum is easily distinguished from the body of the stomach by its mucosa, which is flatter and without rugae. The antrum begins just distal to the termination of the gastric canal.It is also histologically distinct, being without chief or parietal (acid-producing) cells. The margin of the antrum is irregular, but definite. Externally, the antrum is difficult to demarcate

Pylorus

The pylorus is a region of the stomach variously called the pyloric canal, pyloric ring, and pyloric valve. Proximally, it merges into the gastric antrum without a definite external boundary; distally, it ends abruptly at the thin-walled duodenum. At its narrowest point, the luminal diameter

never exceeds 19 mm. The size is important in estimating the optimal size of artificial openings, such as in gastrojejunostomies or pyloroplasties.

At the pyloroduodenal junction, the continuity of the circular musculature is interrupted by an anular septum that arises from the connective tissue of the submucosa. Proximal to this ring, the circular muscle layer is thickened to form the pyloric sphincter. Distal to the ring, the circular muscle coat at the duodenum is thinner. The sudden decrease in wall thickness as one passes from the pylorus to duodenum results in an "os pylorus," surrounded by a duodenal "fornix." The existence of this fornix must be kept in mind when performing pyloromyotomy

The lesser curvature of the antrum, the pylorus, and the upper border of the duodenum are attached to the hepatogastric and hepatoduodenal ligaments (lesser omentum). The greater curvature is attached to the gastrocolic ligament (greater omentum).

Relations of the Distal Gastric Surgical Unit

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Posterior relations:

Floor of lesser sac

Transverse mesocolon

Head and neck of pancreas

Aorta and celiac trunk and its branches

Celiac ganglion and plexus

Hepatic triad

Gastroduodenal artery

Anterior relations:

Anterior abdominal wall

Medial segment of left lobe and anterior segment of right lobe of liver

Transverse mesocolon

Neck of gallbladder (if stomach is empty)

PROXIMAL UNIT:

The proximal gastric unit consists of the distal esophagus, esophageal hiatus, and proximal stomach. The esophagus joins the stomach in the abdomen, just below the diaphragm. The abdominal oesophagus measures about 2.5cm.

Blood supply of Stomach:

Stomach has a rich bloodsupply mainly through celiac plexus.

The four main arteries which has the blood supply will be

1.left gastric- largest branch from celiac trunk

2.Right gastro epiploic – from gastroduodenal artery

3.Left gastroepiploic –branch from splenic artery

4.Right gastric –from hepatic artery

The above mentioned vessels along with others form an anastomotic network which travels to both curvatures of stomach .The left and right gastric artery passes along greater curvature and both the gastro epiploic vessels accompanies along the border of lesser curvature.

Venous drainage: The stomachs venous system has the following veins:

Left Gastric (Coronary) Vein

Right Gastric Vein

Right Gastroepiploic Vein

Left Gastroepiploic Vein

Left Inferior Phrenic Vein

Short Gastric Veins

Lymphatic Drainage

Lymphatics of proximal half of stomach of stomach drains into left gastric, splenic, and superior pancreatic lymph nodes. From antrum it drains into right gastric, right gastroepiploic and subpyloric lymph. Nodes. From the pylorus ,it drains into right gastric and subpyloric nodes.

Efferent lymphatics from suprapyloric region drains into para aortic lymph nodes and into left supraclavicular nodes. Efferent lymphatics from subpyloric lymph nodes into superior mesenteric lymph nodes. Lymphatics near oesophagogastric junction communicates with oesophageal lymphatics. In carcinoma stomach if upper lymphatics blocked, reterograde spread through lower lymphatics occur. In carcinoma stomach different resections are classified as RO,R1,R2.,R3 or D1.D2 Based on the levels of lymph nodes in the abdomen in relation to the stomach.R0 is no residual disease.R1 is microscopic residual disease. R2 is Macroscopic residual disease. R3 is inoperable. Zone 1 lies in gastrocolic omentum along the right gastroepiploic vessels, draining pyloric portion of the greater curve to pyloric, coeliac and aortic lymph nodes.

Zone 2 lies in gastrocolic and gastrosplenic omentum along the left gastoepiploic vessels draining from upper half of greater curve to pancreaticosplenic and aortic lymph nodes.

Zone 3 is drainage from proximal two thirds of the stomach and the upper lesser curve along the left gastric artery. And drains into perioesophageal lymph nodes.

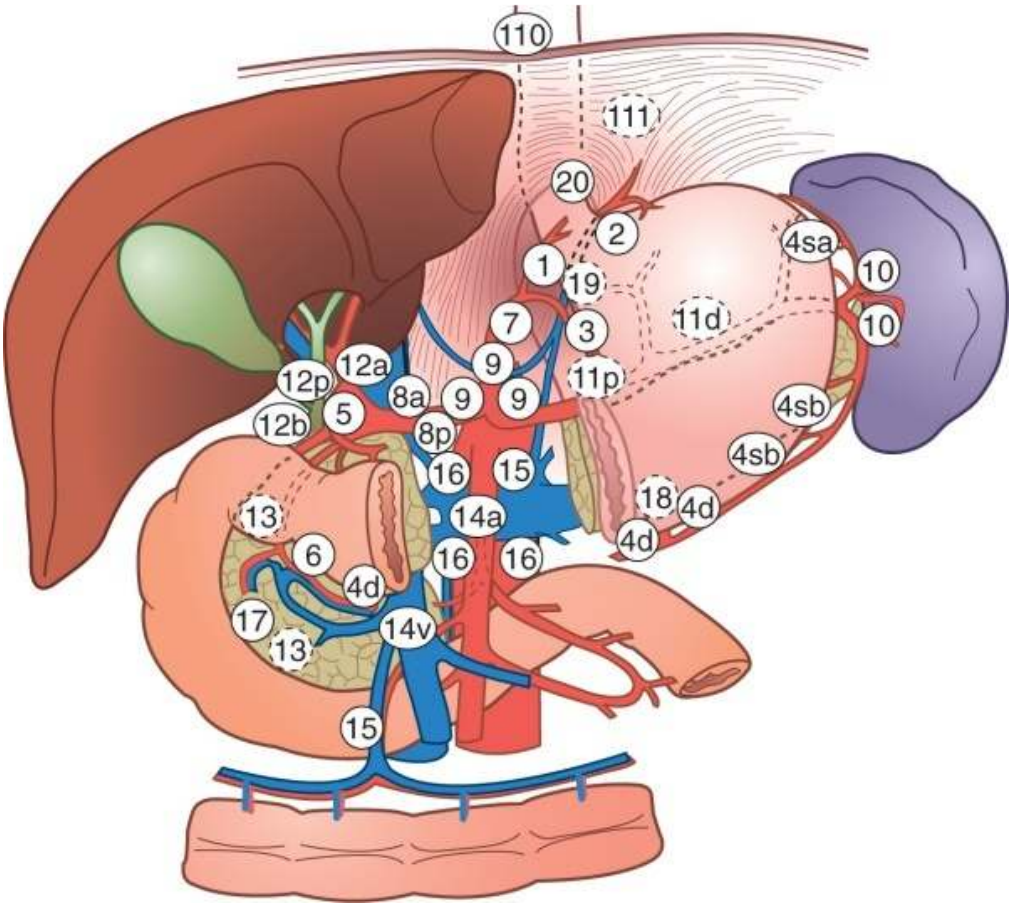
Zone 4 is from distal portion of lesser curve and pylorus along hepatic artery into para aortic lymph nodes.

LYMPH NODE STATION

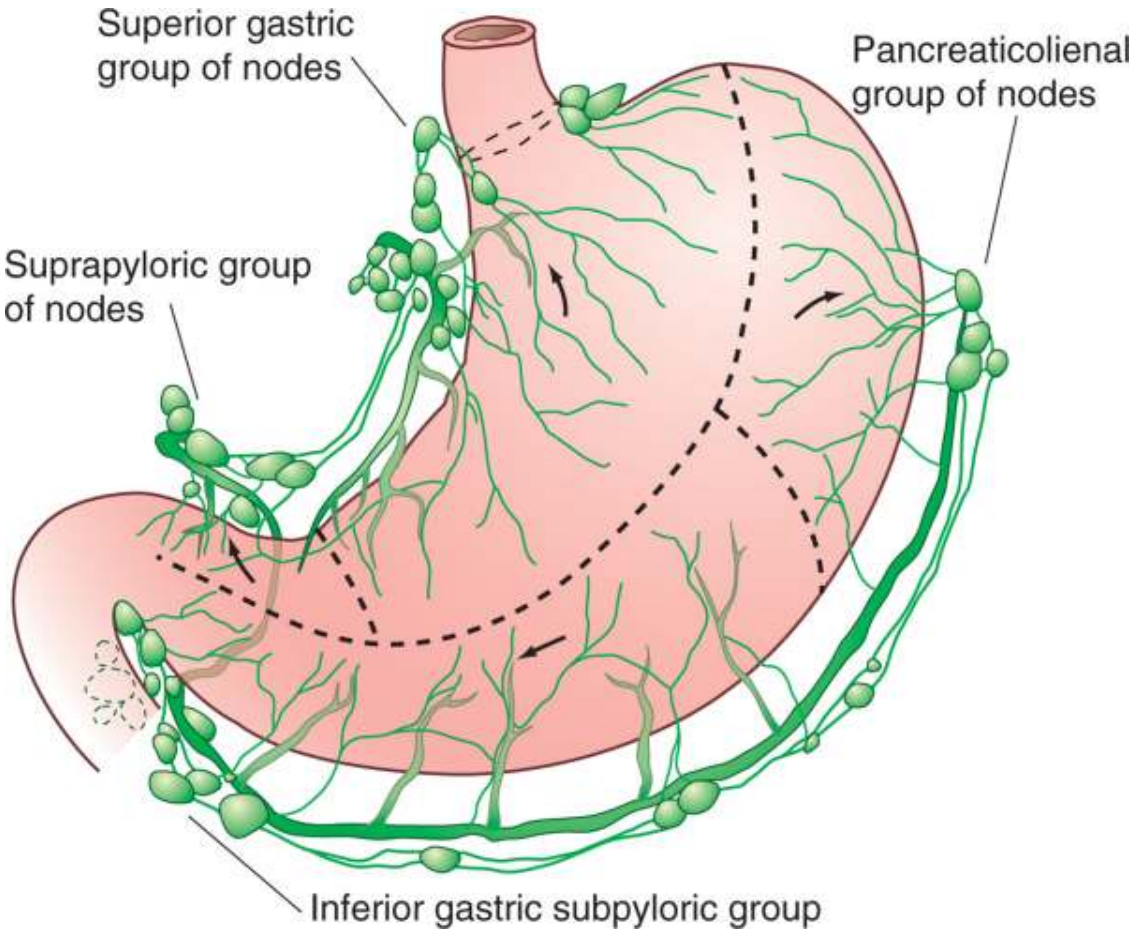
1	Right paracardial
2	Left paracardial
3	Lesser curvature
4sa	Short gastric
4sb	Left gastroepiploic
4d	Right gastroepiploic
5	Suprapyloric
6	Infrapyloric
7	Left gastric artery
8a	Anterior comm. hepatic

8p	Posterior comm. hepatic
9	Celiac artery
10	Splenic hilum
11p	Proximal splenic
11d	Distal splenic
12a	Left hepatoduodenal
12b,p	Posterior hepatoduodenal
13	Retropancreatic
14v	Superior mesenteric vein
14a	Superior mesenteric artery
15	Middle colic
16al	Aortic hiatus
16a2,b1	Para-aortic, middle
16b2	Para-aortic, caudal

Lymphnode Station



Lymphatic Drainage of Stomch



Gastric Innervation

Two nerve plexuses, the submucosal or Meissner and the myenteric or Auerbach plexus, represent "a brain within the gastrointestinal tract." - Elder and Deakin

The above statement was true because stomach has both forms of nerve supply. They are intrinsic and extrinsic pathways .Both are independent of the other and they play pivotal role in both secretory as well as motor functions. Both the right and left vagus nerves provides the external parasympathetic nerve supply of the stomach. The important neurotransmitter is the acetylcholine. The pathway of the vagus nerve starts from the nuclei belongs to the nerve, which is present in the floor of the fourth ventricle. From there, it passes through the carotid sheath in the neck. It enters the mediastinum andhereit gives off recurrent laryngeal nerve. Further it divides into several branches which winds around esophagus, and here only it forms right and left vagus nerves.

Near the Gastro Esophageal junction the anterior vagus sends a branch (or branches) to the liver in the gastrohepatic ligament, and continues along the lesser curvature as the anterior nerve of Latarjet. Similarly, the posterior vagus sends branches to the celiac plexus and continues along the posterior lesser curvature. The nerves of Latarjet send

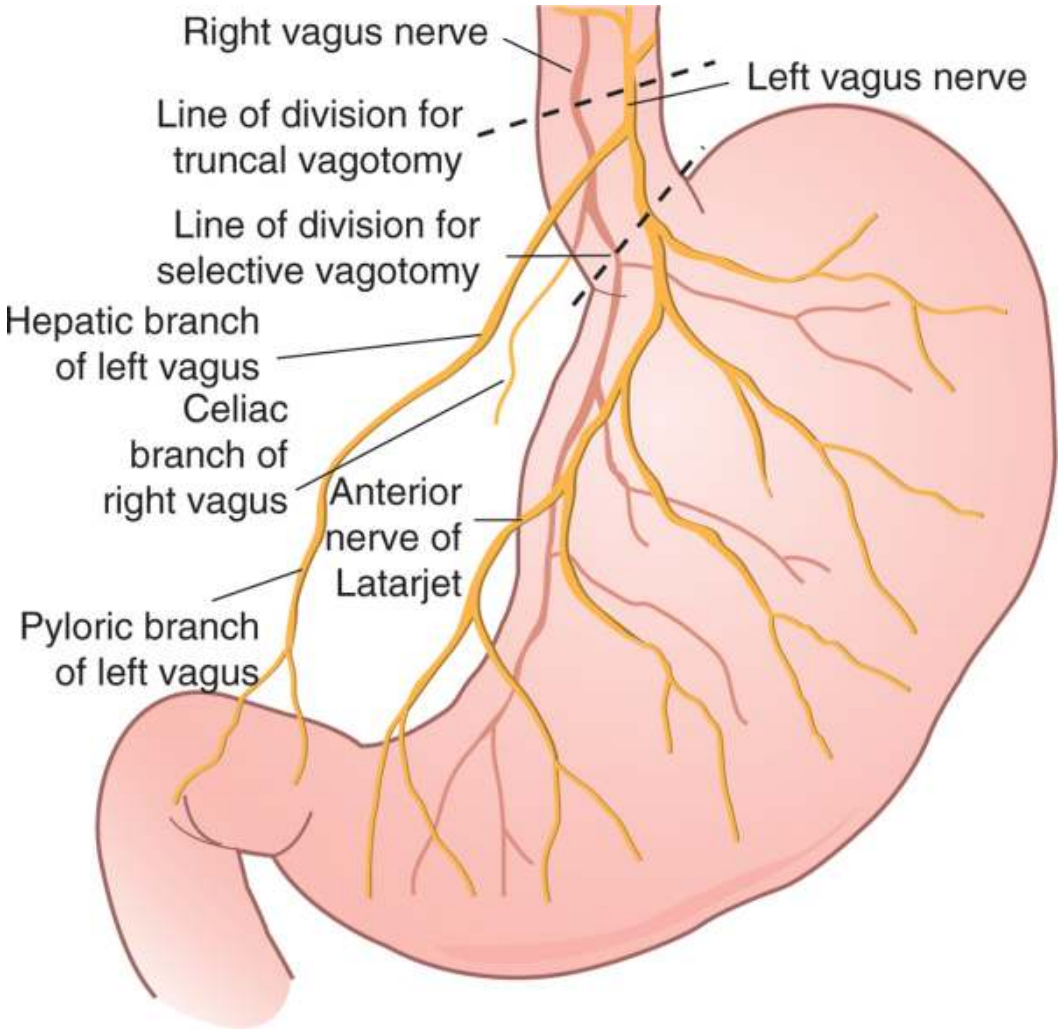
segmental branches to the body of the stomach before they terminate near the angularis incisura as the "crow's foot," sending branches to the antropyloric region.

In 50% of patients, there are more than two vagal nerves at the esophageal hiatus. The branch that the posterior vagus sends to the posterior fundus is termed the criminal nerve of Grassi. This branch typically arises above the esophageal hiatus and is easily missed during truncal or highly selective vagotomy (HSV). Vagal fibers originating in the brain synapse with neurons in Auerbach's myenteric plexus and Meissner's submucosal plexus. Although clinicians are accustomed to thinking about the vagus nerves as important efferent nerves (i.e., carrying stimuli to the viscera), it is important to consider the fact that fully 75% of the axons contained in the vagal trunks are afferent (i.e., carrying stimuli from the viscera to the brain).

The extrinsic sympathetic nerve supply to the stomach originates at spinal levels T5 through T10 and travels in the splanchnic nerves to the celiac ganglion. Postganglionic sympathetic nerves then travel from the celiac ganglion to the stomach along the blood vessels. Neurons in the myenteric and submucosal plexuses constitute the intrinsic nervous system

of the stomach. There may be more intrinsic gastric neurons than extrinsic neurons, but their function is poorly understood.

Nerve Supply of Stomch



Histology

Knowing the histology of the stomach is important because the varius histological types of gastric malignancy plays a role in prognosis. Stomach wall has four vital layers. The first one is

mucosa,then submucosa, latter muscularis propria follows. Lastly the submucosa forms the final layer.

The mucosa has epithelium , lamina propria.and muscularis mucosa.The lining epithelium of the stomachs inner layer is columnar epithelium. This columnar epithelium is of different types.

The lamina propria is an important layer. It lies underneath the basement membrane. It contains blood vessels, along with nerve fibres, inflammatory cells, and few connective tissue.

Next part in the mucosa is muscularis mucosa, which comes under lamina propria. It is a thin mucosal layer and it forms the deepest boundary guts mucosal layer.

The stomach has a epithelial lining of different types, which varies according to the site. The main epithelial lining is with columnar and glandular. When the gastric mucosa was analysed using electro micrograph, it has openings of gastric glands. Various types of epithelial cells will have the lining over the gastric glands.

The main constituents of gastric glands is the endocrine cells. These cells constantly gets denuded and replenished. The important cell type is mucus secreting cells, which are called surface epithelial cells. It is deeply rooted into the gland pits and it secrete

bicarbonate. This secretion of bicarbonate protects the stomach from getting injured by the acids, pepsin and other irritants

Intrinsic Gastric Innervation:

The intrinsic gastric innervations of the stomach is related to its physiological role. Various gastric functions is regulated by hormonal and neural methods. The hormonal mediators controls the stomachs functions by endocrine, paracrine and neurocrine ways.

Gastric acid secretion is mainly regulated by the following enzymes. They are acetlcholine, histamine and gastrin. Among these acetylcholine is the main mediator, which is released through vagal para sympathetic cells

The vagus nerve innervates parietal, G cells and enterochromaffin like cells. Gastrin and cholecystokinin (CCK) receptors are present in parietal cells. Muscarinic receptors and its M3 type acts as a mediator for acetylcholine. Histamine receptor, H 2 subtype produces histamine like effect. The other receptors will be somatostatin receptor and secondary messengers like cyclic AMP and calcium.

Gastric Physiology:

The main gastric functions are gastric acid secretion, maintenance of gastric Ph,gastric motility..Gastric acid secretion is

regulated by acetylcholine, histamine and gastrin. The basal acid secretion is ten percentage of maximal acid output. It is reduced by ninety percentage by after vagotomy or H2 receptor blockage.

The phases of gastric secretion are cephalic phase, gastric phase, and intestinal phase. The cephalic phase is stimulated through smell ,sight, taste and muscarinic receptors. The gastric phase is stimulated through food enters the stomach. The intestinal phase is mediated by chime entering the small bowel.

The luminal gastric pH is 2.The pH at surface epithelial cells is 7.These cells secrete bicarbonate continuously into the lining mucous gel. It keeps the pH of surface mucous at 5.Bicarbonate in mucous barrier reduces luminal acidity.

The functions of gastric acid is conversion of pepsinogen to pepsin. It hydrolyses proteins into polypeptides. It will promote the release of duodenal secretion and prevents bacterial colonisation of upper gastro intestinal tract. It helps formation of food chyme.

The gastric juice contents are hydrochloric acid, mucus, swallowed saliva, reflux content from duodenum. Parietal cells secrete electrolytes and intrinsic factor. It is muco protein.It is essential for absorption of vitamin B12 in ileum. Its secretion is independent of

acid secretion from parietal cells. Pepsinogen is a proteolytic enzyme. There are two types of cells secreted by chief cells. They are type 1 and 2.Type 1 is produced only in stomach. Type 2 is secreted from surface epithelial cells of entire stomach and proximal duodenum. Mucus is secreted by surface mucus cells and mucus neck cells from acid secreting area of stomach and antrum. Gel like mucous contains contains 85% water and 15% glycoproteins. It contains bicarbonate secreted from the surface epithelial cells. Mucus is strong gastric barrier. Mucus secretion is inhibited by anticholinergics. H.pylori break the mucin.

The gastric motility is begins from pace maker cell of cajal. It is located at proximal stomach. Special myoelectric migrating complex slow waves with electric spikes maintain gastric motility in three phases. Immediately after food intake resting tone of fundus and proximal stomach decrease causing receptive relaxation and gastric accommodation mediated by vagus.

Etiology of Gastric Malignancies:

There are umpteen number etiologies postulated with relation to Gastric cancer. Of these environmental, occupational ,food related causes

predominates Others will be smoking and tobacco related causes, overweight, consumption of alchohol contributes to lesser extent.

The risk factors are mentioned below:

1. Low intake of fresh vegetables,fruits comes under dietary habit risk factor
2. Excessive consumption of red meat,smoked foods also a risk factor.
3. Consuming diets which are rich in nitrosoamines and lead.
4. Viral infections like Ebstein Barr infection
5. Economic status also has an impact over site preference Distal site is involved in lower income group and proximal in affluent society.

Some precancerous lesions are mentioned below:

1) Chronic gastritis
2) Patients with pernicious anemia
3) H.pylori infection
4) Polyp of adenomatous orgin which is more than 2cm
5) Patients having benign gastric ulcer, agammaglobulinemia,
6) History of previous gastricsurgery
7) Menetriers disease

Genetic factors:

Most gastric tumours are aneuploid. The familial type was ten percentage. Napolean and many members of his family died of carcinoma stomach. Familial gastric cancer is associated with mutation of E-cadherin gene. It causes hereditary diffuse gastric cancer. Relatives of such family shows mutation of this gene. Inactivation of p53, over expression of growth factors, BCL 2 gene mutation are other genetic causes.

Blood group a:

A ‘is more susceptible for carcinogens. Diffuse type. It is due to different mucopolysaccharide secretion in stomach of blood group A patients.

Ebstein barr virus:

It infects gastric epithelial cells. These tumours have prominent lymphoid stroma.

Environmental and dietary factors:

These factors are of greater importance. Smoked food contains polycyclic hydrocarbons which are carcinogens. The widespread use of refrigeration shows decline in the incidence of tumour in united states.

Among the dietary habits, spicy food along with high intake of salt in food are the major determinants in occurrence of gastric malignancy.

In atrophic gastritis and achlorhydria ,stomach predispose to the production of N-nitroso carcinogens. High gastric p H promote bacterial overgrowth in the stomach. The organisms converte the nitrites to carcinogenic nitrogens. Defect in the mucosal barrier facilitate the penetration of carcinogens. Cigarette smoking ,alcohol have increased risk. Increased concentration of zinc and lead in drinking water have increased risk.

The occupational causes are rubber workers and coal workers. Zinc, lead. talc .and asbestos cause carcinoma stomach. High consumption of fruits have low incidence of gastric cancers. Fruits and vegetables rich in vitamin C protect from carcinoma stomach.

Predisposing Conditions

Polyps

There are five types of gastric epithelial polyps: inflammatory, hamartomatous, heterotopic, hyperplastic, and adenoma. The first three

types have negligible malignant potential. Adenomas can lead to carcinoma, just like in the colon, and should be removed when diagnosed. Occasionally, hyperplastic polyps can be associated with carcinoma. Patients with familial adenomatous polyposis have a high prevalence of gastric adenomatous polyps(about 50%), and are 10 times more likely to develop adenocarcinoma of the stomach than the general population. Screening EGD is indicated in these families. Patients with hereditary nonpolyposis colorectal cancer may also be at risk for gastric cancer.

Atrophic Gastritis

Chronic atrophic gastritis is by far the most common precursor for gastric cancer, particularly the intestinal subtype The prevalence of atrophic gastritis is higher in older age groups, but it is also common in younger people in areas with a high incidence of gastric cancer. In many patients, it is likely that H.pyloriis involved in the pathogenesis of atrophic gastritis. Correa described three distinct patterns of chronicatrophic gastritis: autoimmune (involves the acid-secreting proximal stomach), hypersecretory (involving the distalstomach), and environmental (involving multiple random areas at the junction of the oxyntic and antral mucosa).

Intestinal Metaplasia

Gastric carcinoma often occurs in an area of intestinal metaplasia. Furthermore, an individual's risk of gastriccancer is proportional to the extent of intestinal metaplasia of the gastric mucosa. These observations strongly suggest that intestinal metaplasia is a precursor lesion to gastric cancer. There are different pathologic subtypes ofintestinal metaplasia in the stomach, based upon the histologic and biochemical characteristics of the changedmucosal glands. In the complete type of intestinal metaplasia, the glands are completely lined with goblet cells and intestinal absorptive cells. These cells are indistinguishable histologically and biochemically from their small bowel counterparts, and are not seen in the normal stomach. There is evidence that eradication of H. Pylori infection leads to significant regression of intestinal metaplasia and improvement in atrophic gastritis. Therefore,treatment of H. pyloriinfection is a reasonable recommendation for patients with these pathologic diagnoses and H.pylori infection.

Benign Gastric Ulcer

Although once considered a premalignant condition, it is likely that the older literature was confounded bymistakenly labeling inadequately biopsied ulcers and healing ulcers as "benign," when, in fact, they were

malignantto begin with. It is now generally recognized that all gastric ulcers are cancer until proven otherwise with adequatebiopsy and follow-up. Even today, carcinomas are occasionally found when adequately biopsied "benign" ulcers areresected for nonhealing. It is more than likely that the factors just discussed above are more significant etiologically in the development of gastric cancer than the history of a benign gastric ulcer.

Gastric Remnant Cancer

It has long been recognized that stomach cancer can develop in the gastric remnant, usually years following distalgastrectomy for PUD. The risk is controversial, but the phenomenon is real. Most tumors develop >10 years following the initial operation, and they usually arise in an area of chronic gastritis, metaplasia, and dysplasia. Thisis often near the stoma, but many of these tumors are quite large at presentation, and are equally divided between intestinal and diffuse subtypes. Most cases have been reported following Billroth IIgastroenterostomy, but there also have been cases following Billroth I gastroduodenostomy. Whether simple loop gastrojejunostomy increases apatient's risk of gastric cancer, and whether a Roux-en-Y anastomosis following gastric resection lowers their riskof

gastric cancer is unknown. Stage for stage, the prognosis for gastric stump cancer is similar to proximal gastric cancer.

Other Premalignant States

A mutated E-cadherin gene is associated with hereditary diffuse gastric cancer. Prophylactic total gastrectomyshould be considered. Obviously, a myriad of genetic and environmental factors will affect members of the same family, and up to 10% of gastric cancer cases appear to be familial without a clear-cut genetic diagnosis. First degree relatives of patients with gastric cancer have a two- to threefold increased risk of developing the disease. Patients with hereditary non polyposis colorectal cancer have a 10% risk of developing gastric cancer, predominantly the intestinal subtype. The mucous cell hyperplasia of Mntrier's disease is generally considered to carry a 5 to 10% risk of adenocarcinoma. Periodic surveillance EGD is prudent in all the above conditions. The glandular hyperplasia associated with gastrinoma is not premalignant, but ECL hyperplasia and/or carcinoid tumors can occur.

Pathology:

The main pathological changes found in carcinoma stomach are dysplasia, which then leads onto early gastric cancer. It is a well known fact that gastric dysplasia is a forerunner of malignancy. Patients with severe dysplastic changes should be considered for gastric resection, if the

abnormality is wide spread or multifocal. Patients with mild dysplasia should be followed with endoscopic surveillance and Helicobacter eradication.

Early Gastric Cancer:

Early gastric is defined as adenocarcinoma which is confined to the mucosa and submucosa, regardless of lymphnode status. Varioous references states that approximately 10% of patients with gastric cancer will have lymph node metastases. Regarding types,there are several types and subtypes based on morphology and histology. The Japanese classification on early gastric cancer is based on morphology. They are protruded, superficial and excavated. Approximately 70% of early gastric cancer are well differentiated and remaining 30% are poorly differentiated

Other morphological and histological subtypes:

The main types of gastric malignancy with relation to morphology will be -polypoid

-fungating

-ulcerstive

-scirrhous

In the first two, bulk of the tumor mass intraluminal. Polypoid tumors are not ulcerated, fungating tumors are elevated intraluminally. In the later two gross subtypes, the bulk of the tumor mass is in the wall of the stomach. Ulcerative masses closely resembles ulcers. Scirrhous type penetrates the full thickness of the stomach. Scirrhous tumors or Linitis plastica have a poor prognosis.

Histological types:

The histological classification in gastric malignancies are important,in relation to prognosis. There are many types based on histology. Of these Laurens classification is commonly used. It classifies gastric cancer into three types intestinal, diffuse and unclassified. The intestinal type which accounts for about 53%,has got favourable prognosis.

This type usually associated with chronic atrophic gastritis, severe intestinal metaplasia and dysplasia, tends to be less aggressive than the diffuse type. The diffuse type which is of 33%,has poor prognosis are common young females. This type is usually poorly differentiated with early gastric wall invasion, also having both submucosal and subserosal lymphatic spread.The last one ,unclassified type tends to be less aggressive than diffuse one.

WHO classification based on histology:

This classification is based upon histological types of gatric carcinoma.

1. Adenocarcinoma -Most common
    -papillary adenocarcinoma
    -Tubular adenocarcinoma
    -Mucinous adenocarcinoma
    -Signet ring cell carcinoma
2. Adenosquamous carcinoma
3. Squamous cell carcinoma
4. Undifferentiated carcinoma
5. Unclassified carcinoma

Other classifications commonly used:

Siewert classification;

Malignancies involving proximal gastric adenocarcinoma are divided into three types

Type 1: Carcinoma in Barrets oesophagus or true esophageal carcinoma extending to gastroesophageal junction. Total esophagectomy with gastric pull through to the neck is needed.

Type 2 : Tumor within 2cm of squamo columnar junction. Total gastrectomy with necessary anastomosis is needed.

Type3 : Tumor in subcardial region.

Borrmann"s Classification:

This is based upon morphological features of tumor

1.Single,polypoid carcinoma

2.Ulcerated carcinoma with clear cut margin

3.Ulcerated margin without clear cut margin

4.Diffuse carcinoma

5.Unclassified

Japanese classification of gastric carcinoma:

This classification is based on the following rules.

-The basic rules for clinical, surgical, pathological and final features.

-Specific rules for histology.

-Gastric biopsy specimen group classification

-Based on chemo radiotherapy response

Nodal spread as per this classification:

N x- nodes unknown/cannot be assessed

N o- no nodal spread

N 1 –Group 1 nodes involved

N 2 -Group 2 nodes involved

N 3 – Group 3 nodes involved

Birmingham staging:

1. confined to mucosa/muscularis propria

2 . Muscularis/serosal involvement

3 –Nodal spread

4a—Residual disease

4b- Metastatic disease

Types of spread in Gastric malignancy:

Gastric cancer spread occurs in multimodal fashion. The metastasis mainly passes through lymphatics, via blood and along transperitonal route.

Lymphatic route:

This spread occurs by permeation and embolisation through lymphatics to subpyloric, gastric, pancreaticoduodenal, splenic, celiac, aortic and lastly to left supraclavicular nodes. Retrograde spread to mesenteric nodes in small and large bowel, signifies poor prognosis.

Blood spread:

This spread occurs to liver causing multiple liver secondaries which are presented as multiplr, hard nodules with umbilications due central necrosis.

Transperitoneal route:

This mode of spread can cause peritoneal seedlings leading onto ascites. Krukenberg tumor in ovary, is the presentation due to this type commonly seen in menstruating women. Rectal secondaries or Blumers shelf, Sister Mary Joseph type of umbilical secondaries occurs through this route.

TNM STAGING

T: Primary tumor

Tis Carcinoma in situ; intraepithelial tumor without invasion of lamina propria

T1 Tumor invades lamina propria or submucosa

T2 Tumor invades muscularis propria or subserosa

T3 Tumor penetrates serosa (visceral peritoneum) without invasion of adjacent structures

T4 Tumor invades adjacent structures

N: Regional lymph node

N0 No regional lymph node metastasis

N1 Metastasis in 1 to 6 regional lymph nodes

N2 Metastasis in 7 to 15 lymph nodes

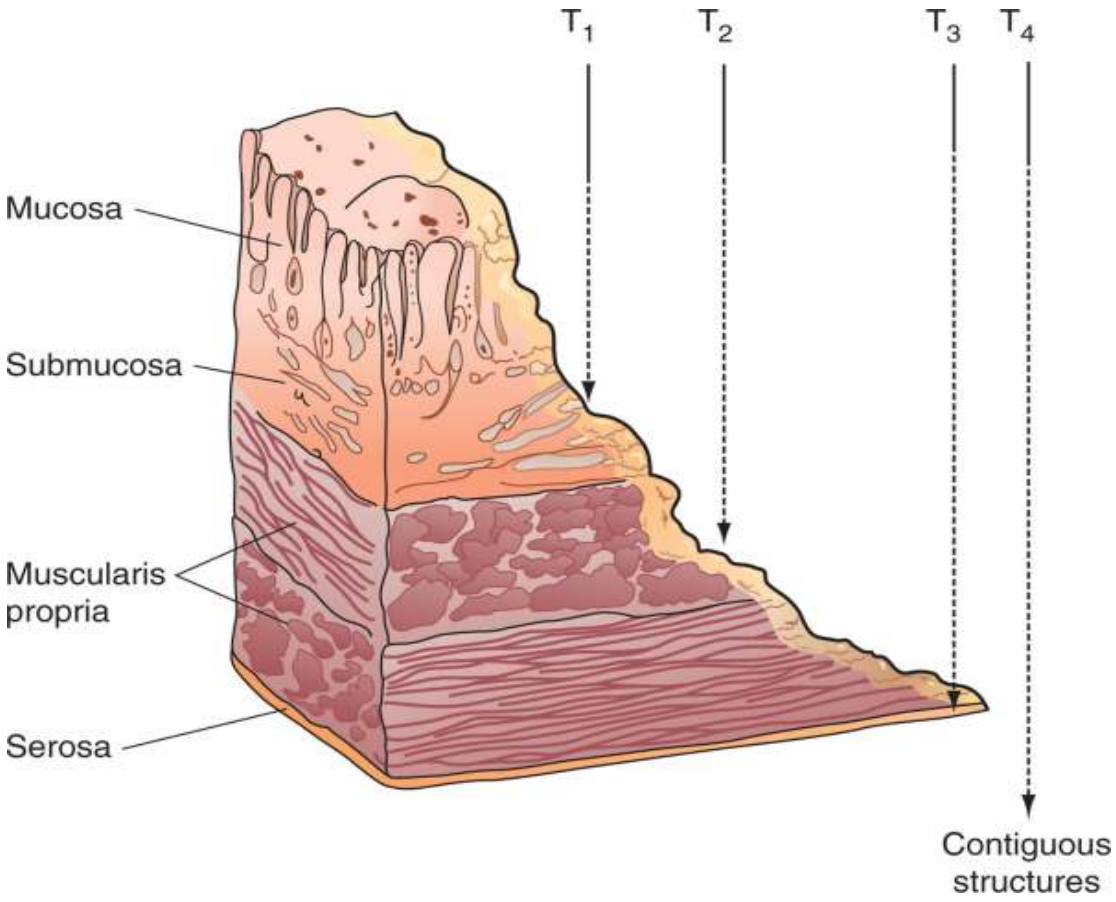
N3 Metastasis in more than 15 regional lymph nodes

M: Distant metastasis

M0 No distant metastasis

M1 Distant metastasis

TNM Staging


T1
T2
T3
T4
Mucosa
Submucosa
Muscularis propria
Serosa
Contiguous structures

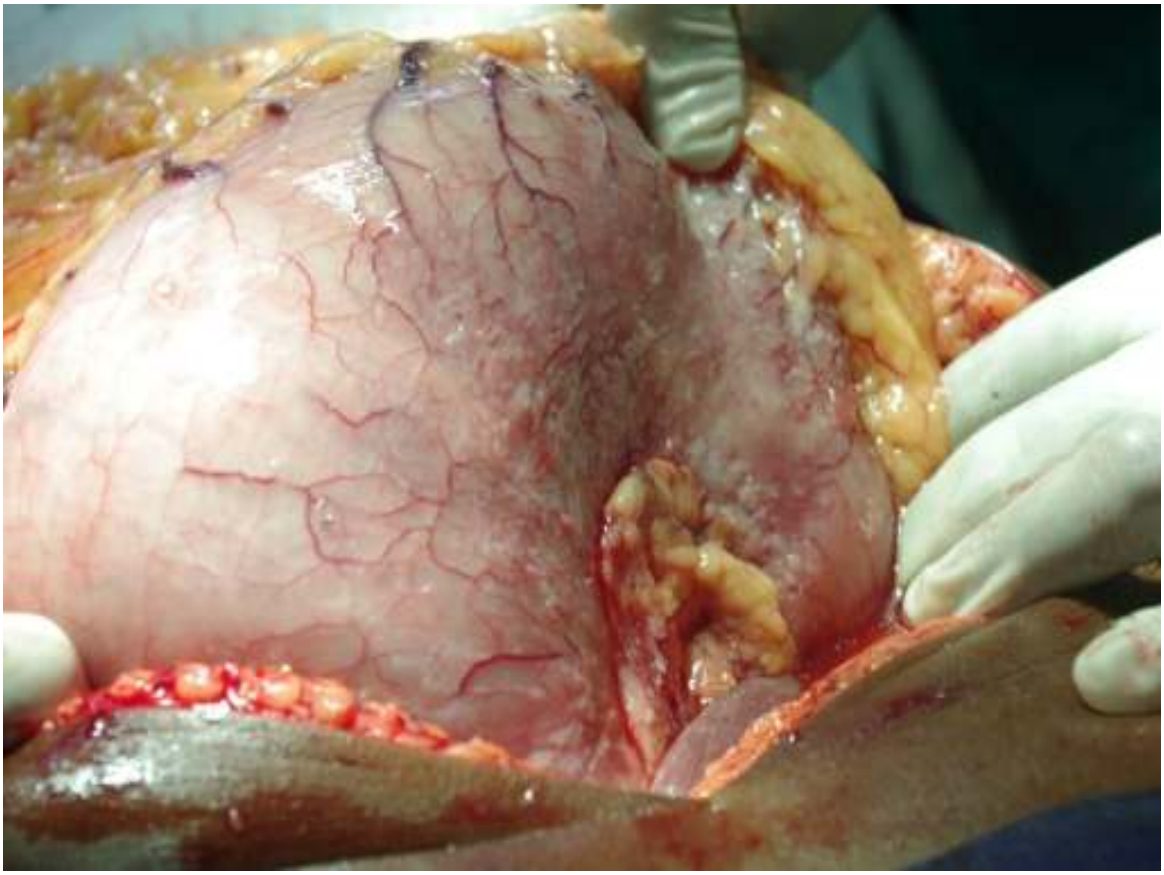
STAGE GROUPING

STAGE	T	N	M
0	Tis	N0	M0
IA	T1	N0	M0
IB	T1	N1	M0
	T2	N0	M0
II	T1	N2	M0
	T2	N1	M0
	T3	N0	M0
IIIA	T2	N2	M0
	T3	N1	M0
	T4	N0	M0
IIIB	T3	N2	M0
IV	T4	N1–3	M0
	T1–3	N3	M0
	Any T	Any N	M1

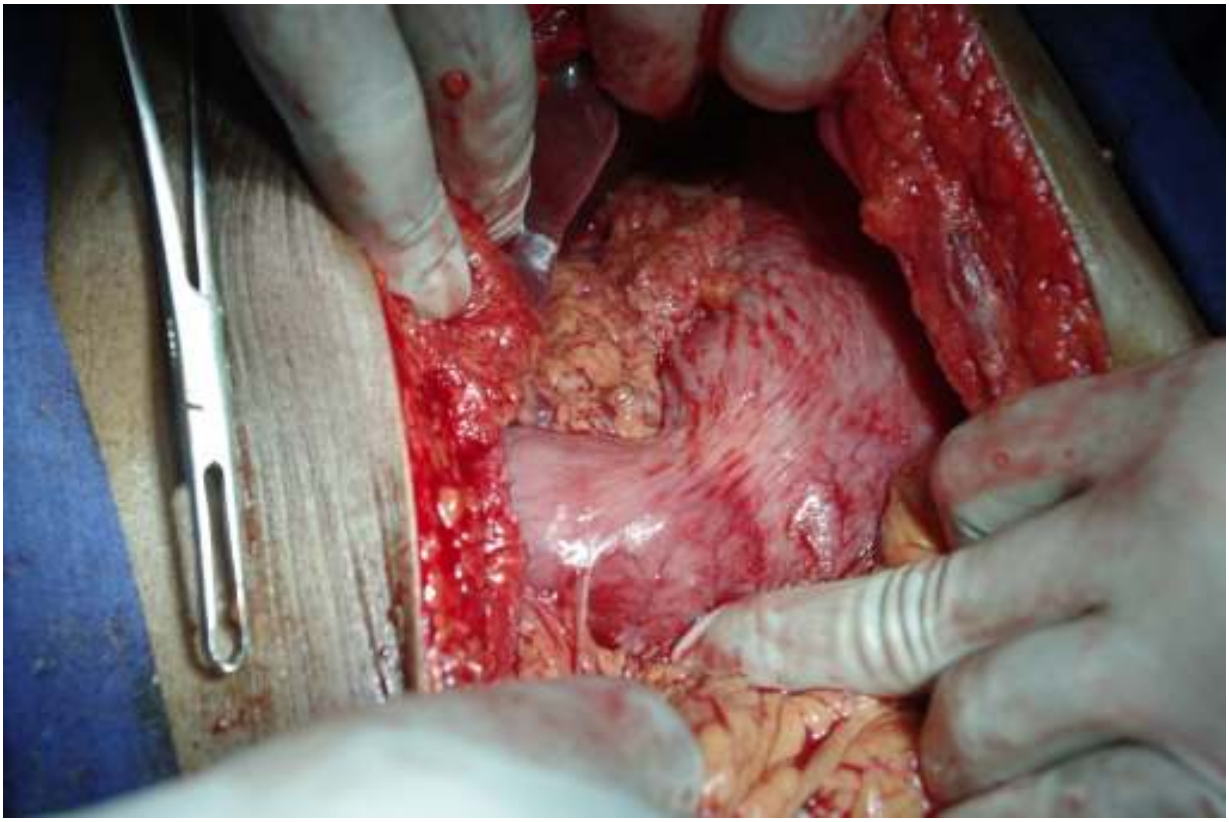
A case of carcinoma stomach presenting as Mass abdomen with Abdominal Distention

The same case with Peritoneal and Omental Deposits

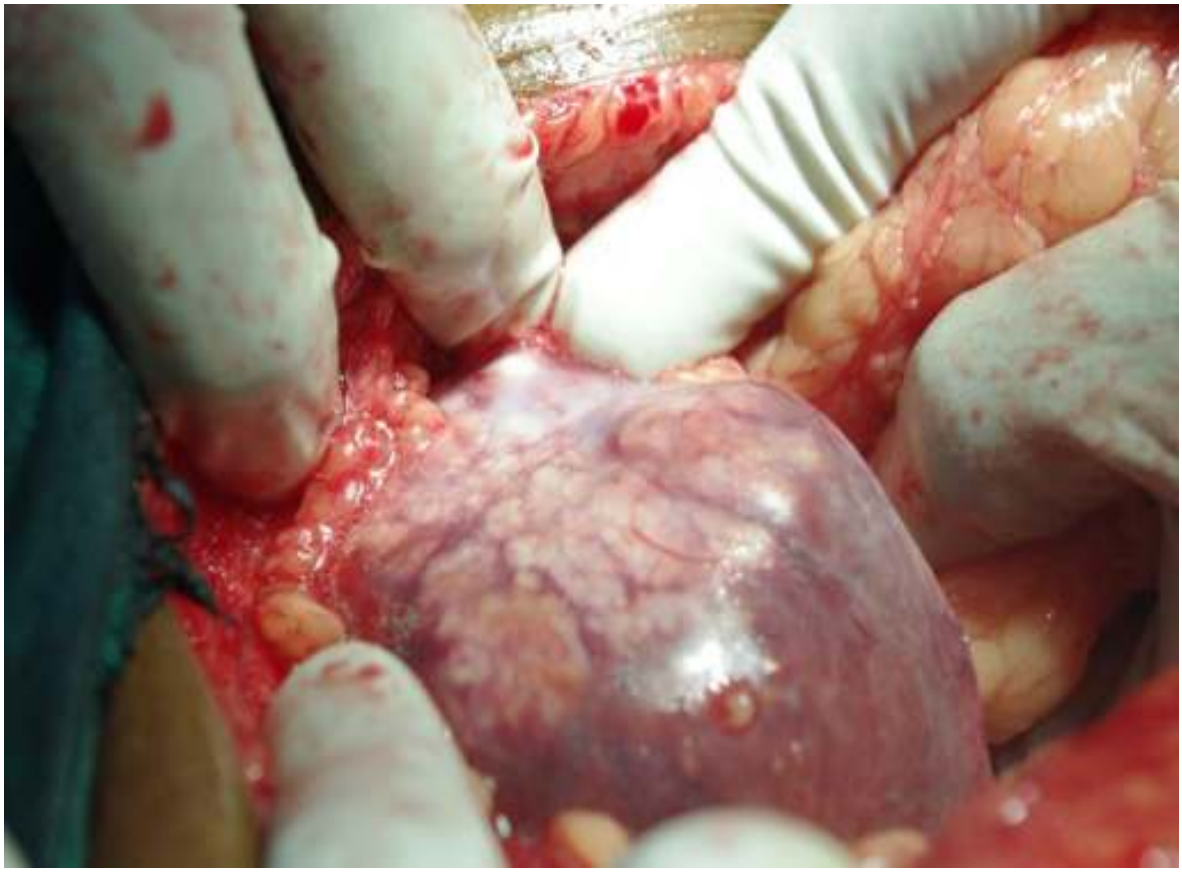
Carcinoma stomach at Cardia and Fundus



A case of Linitis Plastica



Liver secondaries in a case of carcinoma stomach



PRESENTATION OF CARCINOMA STOMACH;

In early gastric cancer patients are, mostly asymptomatic. The non specific symptoms are indigestion, vague epigastric discomfort, constant radiating pain which is not related to food intake.The specific symptoms depend on the site of the tumour. It includes obstruction, dysphagia, and mass in metastatic disease, liver secondraies, ascites, secondries in ovary, rectovesical pouch, and umbilicus. The unusual presentations are acanthosis nigricans,Irish nodes in the axilla.

CLINICAL MANIFESTATIONS:

The most common manifestations are recent onset of loss of weight and appetite, early satiety and fatigue. The most common sign is anaemia. It is of microcytic hypochromic anaemia, and mostly due to iron deficiency and it occurs in forty percentage of presentation. The other features are upper abdominal pain, vomiting with features of gastric outlet obstruction. In advanced cases most of the patients were present with mass abdomen. The mass in pylorus lies above the umbilicus, nodular, hard, with impaired resonance, mobile , and it moves with respiration, all borders are well made out..When it arises from the body of the stomach, it present as only mass abdomen..The

presence of epigastric mass is a poor prognostic sign.10% of patients were present with palpable cervical nodes, ascites, jaundice. Sister josephs nodule is a visible and palpable secondary deposit at the umbilicus. It is due to spread along the lymphatics around the falciform ligament. It is a poor prognostic sign. Trosiers sign is an enlarged lympnode in the left supraclavicular fossa. It indicates lymphatic spread via thoracic duct.

Diagnostic evaluation

Distinguishing between peptic ulcer and gastric cancer on clinical grounds alone is usually impossible. Patients who were above the age of 45 years and having new-onset dyspepsia, as well as all patients with dyspepsia and alarm symptoms (weight loss, recurrent vomiting, dysphagia, evidence of bleeding, or anemia) or with a family history of gastric cancer should have prompt upper endoscopy and biopsy if a mucosal lesion is noted. Essentially, all patients in whom gastric cancer is part of the differential diagnosis should have endoscopy and biopsy.

If suspicion for cancer is high and the biopsy is negative, the patient should be re-endoscoped and more aggressively biopsied. In some patients with gastric tumors, upper GI series can be helpful in planning treatment.

Although a good double-contrast barium upper GI examination is sensitive for gastric tumors (up to 75% sensitive), in most centers, endoscopy has become the gold standard for the diagnosis of Gastric malignancy.

Preoperative staging of gastric cancer is best accomplished with abdominal/pelvic CT scanning with IV and oral contrast. MRI is probably comparable. The best way to stage the tumor locally is via EUS, which gives fairly accurate (80%) information about the depth of tumor penetration into the gastric wall, and can usually show enlarged (>5 mm) perigastric and celiac lymph nodes. In some centers, if the tumor is transmural (T3) or involves lymph nodes (enlarged nodes can usually be needled under ultrasound guidance), preoperative (neoadjuvant) chemotherapy is given. However, there are limitations to tumor staging with EUS. It largely is operator dependent and may underestimate lymph node involvement because normal-sized nodes (<5 mm) can harbour metastases.EUS is most accurate in distinguishing early gastric cancer (T1) from more advanced tumors.

Positron Emission Tomography Scanning

Whole-body PET scanning uses the principle that tumor cells preferentially accumulate positron-emitting 18 Ffluorodeoxyglucose. This modality is most useful in the evaluation of distant metastasis in gastric

cancer but cannot so be useful in loco regional staging. PET scan is most useful when combined with spiral CT (PET-CT) and should be considered before major surgery in patients with particularly high-risk tumors or multiple medical co morbidities.

Staging laparoscopy and peritoneal cytology

To some extent, the usefulness of these modalities depends on the individual patient's situation as well as the treatment philosophy of the cancer team. The fundamental question is "will it make a difference to this patient's management?" Patients with gastric cancer who undergo R0 resection (i.e., no gross residual disease) and are found to have positive peritoneal cytology (no gross carcinomatosis) have a much worse prognosis than thecytology negative group . It is controversial how much this information adds prognostically to that of pathologic staging (TNM). Whether this poor prognosis can be improved postresection with aggressive adjuvant treatment (systemic, or local intraperitoneal hyperthermic chemotherapy)is unknown. Unfortunately, it is also unclear how much these patients benefit from gastric resection.

Currently peritoneal cytology information is unlikely to change the treatment of patients with gastric cancer, and most patients without detectable distant metastases will have (and should have) gastric resection

regardless of the peritoneal cytology results. A quick laparoscopic examination can occasionally reveal small peritoneal implants orliver metastases that were not detected on preoperative imaging studies and, in some patients (e.g., high risk for surgery or impressive carcinomatosis), this will change the operative plan and avoid a major but futile surgical procedure. Laparoscopy may be most useful in patients with proximal tumors or with adenopathy on spiral CT scan. An extensive laparoscopic staging procedure, although quite accurate, has not been widely adopted.

Treatment

Surgical removal of the tumor is the main stay as the treatment in most of the patients. With clinically resectable local and regional disease, gastric resection should be considered.. Obvious exceptions include patients will be patients who cannot bear the major abdominal resection, and presentation in late stage along with extensive metastasis. The aim of curative surgical treatment is resection of all tumor (i.e., R0 resection). Thus, all margins should be free and extensive and needed lymph node excision should done.

Generally, the surgeon strives for a grossly negative margin of at least 5 cm. Some gastric tumors, particularly the diffuse variety, are quite infiltrative and tumor cells can extend well beyond the tumor mass; thus,

gross margins beyond 5 cm may be desirable. Frozen section confirmation of negative margins is important when performing operation for cure, but it is less important in patients with nodal metastases beyond the N1 nodal basin.

It should be strongly emphasized that many patients with positive lymph nodes are cured by adequate surgery. It should also be stressed that often lymph nodes that appear to be grossly involved with tumor turn out to be benign or reactive on pathologic examination. More than 15 resected lymph nodes are required for adequate staging. Therapeutic nihilism should be avoided and, in the low-risk patient, an aggressive attempt to resect all tumor should be made. The primary tumor may be resected enbloc with adjacent involved organs (e.g., distal pancreas, transverse colon, or spleen)during the course of curative gastrectomy. Palliative gastrectomy may be indicated in some patients with obviously incurable disease, but most patients presenting with stage IV gastric cancer can be managed without major operation.

Extent of Gastrectomy

The extent of gastric resection, is quite debatable issue. But radical subtotal gastrectomy still considered as gold standard. Unless required for R0 resection, total gastrectomy confers no additional survival benefit and may have adverse nutritional or quality-of-life consequences, and higher peri operative morbidity and mortality. Subtotal gastric resection typically involves .the following important procedures- ligation of vessels like gastric arteries,epiploic arteries at level of its orgin, Next is total removal tumor involved part which very often comes around major part of distal stomach, two cm of duodenum and removal of greater and lesser omentum, along with the exicision all involved lymph nodes. After this Billroth type 2 gastrojejunostomy is performed.

Radical subtotal gastrectomy is generally deemed to be an adequate cancer operation in most Western countries, provided that the contingencies stated result in tumor-free margins, >15 lymph nodes, and the resection of all gross tumor. In the absence of involvement by direct extension, the spleen and pancreatic tail are not removed.

Total gastrectomy with Roux-en-Y esophagojejunostomy may be required for R0 resection and maybe the best operation for patients with proximal gastric adenocarcinoma. The construction of a jejunal pouch maybe beneficial nutritionally, particularly for those patients with a good prognosis. Proximal subtotal gastric resection, a technically feasible alternative to total gastrectomy for some proximal gastric tumors, requires an esophago gastrostomy to a vagotomized distal gastric remnant. Pyloroplasty in this setting virtually guarantees bile esophagitis, and if the pylorus is left intact, gastric emptying may be problematic. An interposition of isoperistalic jejunum between the esophagus and antrum

could be considered as an alternative reconstruction, but, all things considered, total gastrectomy usually results in superior functional, if not oncologic,results for most patients with proximal gastric cancer.

Extent of Lymphadenectomy

The Japanese Research Society for Gastric Cancer has numbered the lymph node stations that potentially drain the stomach. Generally these are grouped into level D1 (i.e., stations 3 to 6), level D2 (i.e., stations 1,2, 7, 8, and 11), and level D3 (i.e., stations 9, 10, and 12) nodes. Generally, D1 nodes are perigastric, D2 nodesare along the hepatic and splenic arteries, and D3 nodes are the most distant. The operation described above (radical subtotal gastrectomy in the Extent of Gastrectomy section), which is by far the most commonly perform edprocedure in the United States for gastric cancer, is called a D1 resection because it removes the tumor and the perigastric D1 nodes. The standard operation for gastric cancer in Asia and specialized U.S. centers is the D2gastrectomy, which involves a more extensive lymphadenectomy (removal of the D1 and D2 nodes).

In addition to the tissue removed in a D1 resection, the standard D2 gastrectomy removes the peritoneal layer over the anteriormesocolon and selectively over the pancreas, along with nodes along the hepatic and splenic arteries, and thecrural nodes. Splenectomy and distal pancreatectomy are not routinely performed, because this clearly has beenshown to increase the morbidity of the operation. Unfortunately, the randomized prospective trials that have been performed have not

confirmed this survival advantage, but the morbidity and mortality in the D2 group was higher. This was mostly attributable to the splenectomy and distal pancreatectomy, which are no longer routinely done as part of the D2 gastrectomy.

Palliative Treatment

Patients with terminal illness needs better care,than cure to improve their quality of life. This will be the main aim of palliative treatment. The incidence of stage 4 disease varies, but often it falls between 20 to 30%.So all modalities of palliative treatment should be considered, before starting the care of the patients. It should be aimed to have minimal morbitity, so that patient can tolerate the treatment. this includes both medical and surgical therapy. Pain relief and symptomatic treatment is the main stay in medical management.

Surgical interventions includes various bypass procedures, feeding jejunostomy. This can be done either by open type or through non operative type. Laser recannulization, endoscopic dilatation along with stent placement, are some examples of this type.

Adjuvant Therapy

Adjuvant therapy is mainly based on chemo and radiotherapy. There are umpteen number of drugs available, but still 5 flurouracil is considered as gold standard. There are lot of trials involving various combination of drugs. So it has be tailor made for the patints and more importantly it should be affordable to the patients. Radiotherapy can be given alone ,or it can be combined along with chemotherapy.

Early complications

1. Any upper abdominal surgery will have effect on cardiovascular system, respiratory system.
2. Anastomotic leak
3. Fluid collection or abscess formation. This occurs if lymph node dissection was done.
4. If drainage of gastric remanent gets blocked,it lead on to vomiting
5. Distruption of duodenal stump occurs, if afferent loop is obstructed.

Late complications:

Surgical resection of gastric malignancy will have major impact on the stomachs normal function.Both anatomical and physiological alterations affects the normal lifestyle of the patients.The commonly noted late complications are reflux gastritis,Dumping syndrome, and nutritional changes.

Reflux gastritis:

This is mainly due to alteration in the pyloric architecture. The biliary and pancreatic contents easily travels across the stomach, there by causing gastritis, since these contents are alkaline.

Medical treatment has little benefits .Some patients may require diversion of the small bowel contents.

-Dumping syndromes:

The symptoms related to this syndrome are vomiting ,loose stools, abdominal pain and fullness .These clinical manifestations may due to hastened gastric emptying. Late dumping symptoms may de due to increase in insulin after food

-Nutritional deficiencies:

Among this iron and vitamin B12 deficiencies are common, because intrinsicfactor which is important for absorption of vitamin B12 will be absent after gastric resection.

Immunotheraphy as an adjuvant:

It is given in stage 3 carcinoma after total gastrectomy. It is starts from 5th postoperative day to the end of 2 years. It is based on preoperative cell counts. Which can be improved/modulated by this therapy. Residual cancer cells or micrometastases can be eradicated by this therapy. Regime contains; initially immunotherapy using weekly intra muscular injections of picibanil (streptococcus pyogenes derived) immune agent 1 clinical unit is given on 5th postoperative day. from 10th postoperative day onwards, chemotherapy regime consisting of mitomycin C and 5 Fu. Mitomycin C 4mg/50 kg IV Twice weekly for two weeks, then weekly for six weeks. 5 FU as 500mg/50jg IV twice weekly for two weeks, then weekly for six weeks, then 600mg/50 kg orally daily b for two years. Neoadjuvent therapy: This can be given to downstage the tumor and surgery can be done later. The main combination of drugs will be ECF-epirubicin, cisplatin, 5-flurouracil. Then EAP (epirubicin, adriamycin, cisplatin) and

FDT (flurouracil, doxorubicin, triazinate) are also used. Newer drugs like Bevacizumab are under trial.

Recent advances in treatment: Although early detection of gastric cancer is possible, it requires cumbersome procedures like upper gastrointestinal scopy. But in Japan screening for high risk population revealed excellent results. So patients in our country most of the time patient are presented with advanced disease. The recent advancement in screening of malignancies related to stomach will be

1. Serial analysis on gene expression
2. Photoflurography
3. serum pepsinogen estimation.

Role of Biological treatment in Gastric Carcinoma:

Monoclononal antibodies are the newer arrival in the treatment for any cancer patients.Their targets will be the genes responsible for carcinogenetic changes.Number of drugs has passed the trial and are in use.Japanese are the pioneers in this type of studies.The commonly used drugs for gastric carcinoma will be Trastuzumab and Bevacizumab.Both these drugs can be given along with chemotherapy for the betterment of patients quality of life after major gastric surgery.Trastuzumab specifically attack the HER-2 protein,there by cuts the genetic abnormal expression.Acute infusion reaction,left heart failure and the cost of the drug arethe major limitations for its routine use.Bevacizumab is an immunogloblulin G1It specifically binds vascular endothelial growth factor to attain its action.

METHODOLOGY

This is prospective study for EVALUATION OF CASES WITH CARCINOMA STOMACH in Tirunelveli Medical College Hospital, Tirunelveli during the period of 18 months, from Feb 2011 to July 2012.. Formal ethical committee approval was obtained. Patients were informed about the study and consent was obtained.

SOURCE OF DATA:

Patients admitted in the Department of General Surgery, Tirunelveli Medical College who were diagnosed as carcinoma stomach.

Design of study:

Prospective study

Period of study:

18 months

Sample size:

103 cases of carcinoma stomach

Stastical method:

Our data is represented in tabulation, graphs

Method of Collection of Data

The present study was, made out from 103 patients of carcinoma stomach admitted in Tirunelveli medical college and hospital during period of Feb 2011 – July2012.After obtaining an informed written consent, clinical history, detailed examination and relevant investigations were done. After confirming the diagnosis, evaluating the nature of the disease and considering the patient general conditions ,treatment planned including both surgical and non surgical measures . If needed secondary analysis of medical records of these patient will be referred if required. The pathology report of the patient are been followed up with an earnest attempt to study all the cases in detail and to profile my observation made out of them.

Inclusion criteria:

1. Male and females with complaints and investigations suggestive of carcinoma stomach.
2. Both operable and inoperable cases.
3. Advanced cases

Collobrating Department: Pathology

RESULTS

Sex Distribution

Gastric cancer is more common in males. 59% of the cases being males in the study. Male female ratio was 3:2

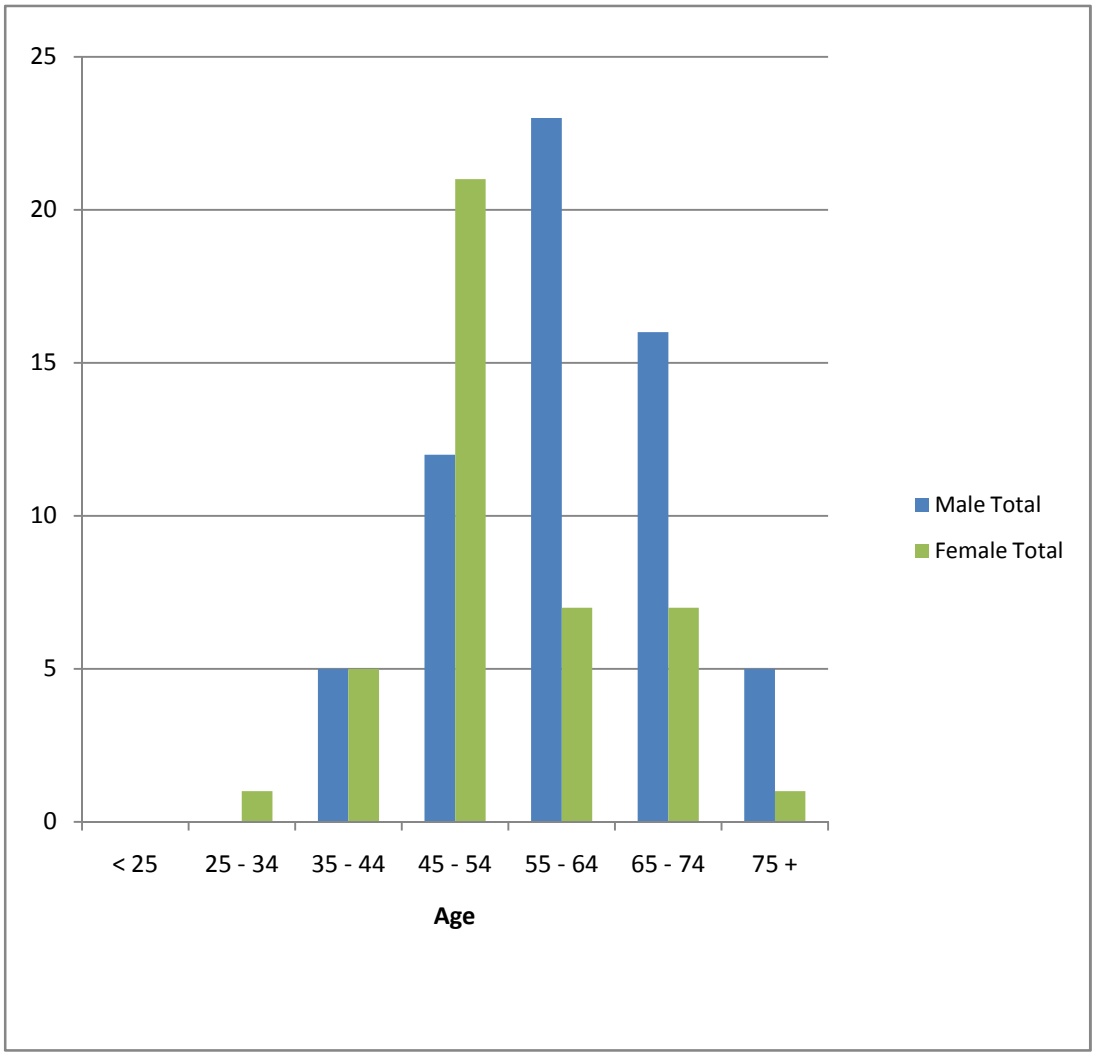
Sex	Total	Percentage %
Male	61	59
Female	42	41

Age Distribution

Carcinoma stomach was more common in the age group of 45 -54 yrs. Increase in incidence was found above the age of 44 years. The youngest patient was aged 25 yrs and oldest was 76 yrs old.

Age	Male		Female	
	Total	%	Total	%
< 25	0	0.0	0	0.0
25 – 34	0	0.0	1	2.4
35 – 44	5	8.2	5	11.9
45 – 54	12	19.7	21	50.0
55 – 64	23	37.7	7	16.7
65 – 74	16	26.2	7	16.7
75 +	5	8.2	1	2.4
Total	61	100.0	42	100.0

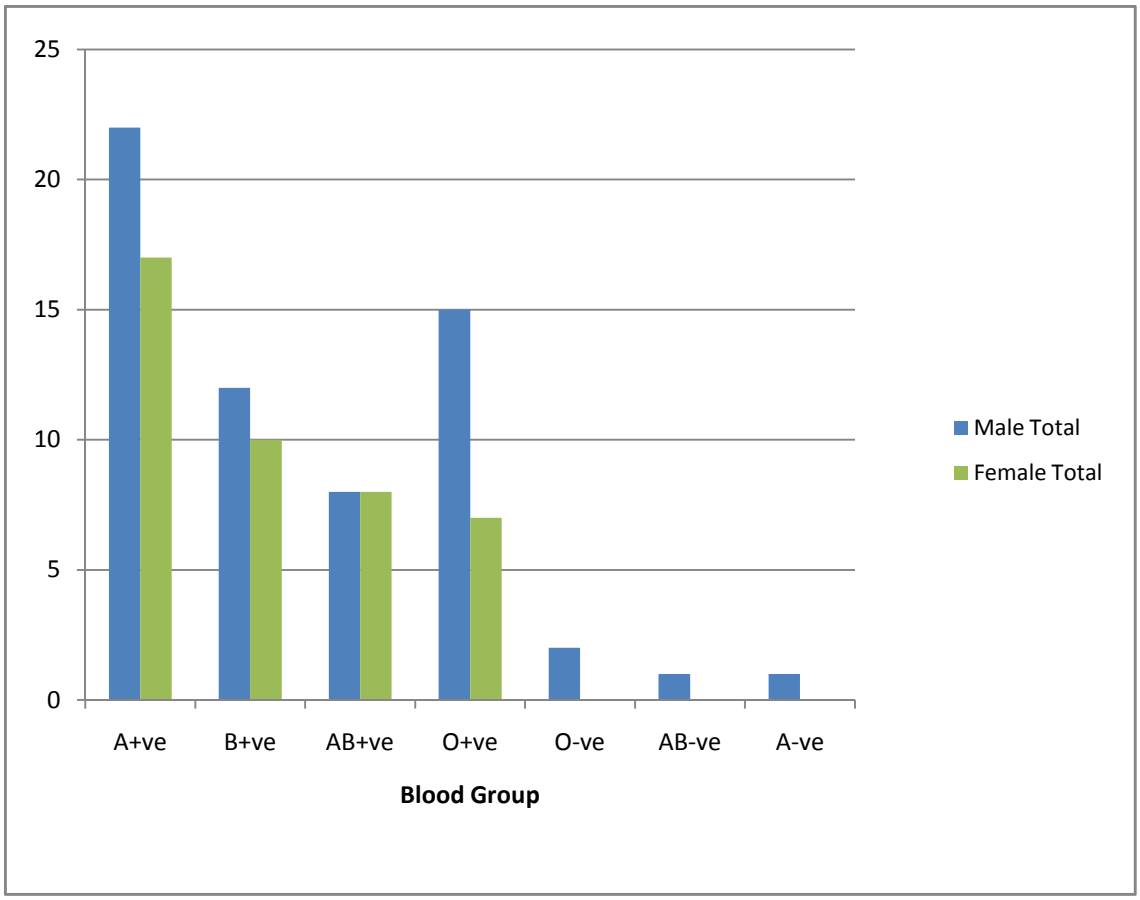
Age Distribution



Blood Group Distribution

Blood group A positive showed the highest association both in males and female. A percentage of 36% in males and 40% in females. Next common blood groups accounted for the occurrence of gastric carcinoma in this study was O positive, B positive. In this study all the Rh negative groups found to be on the lower side, which was shown on below table.

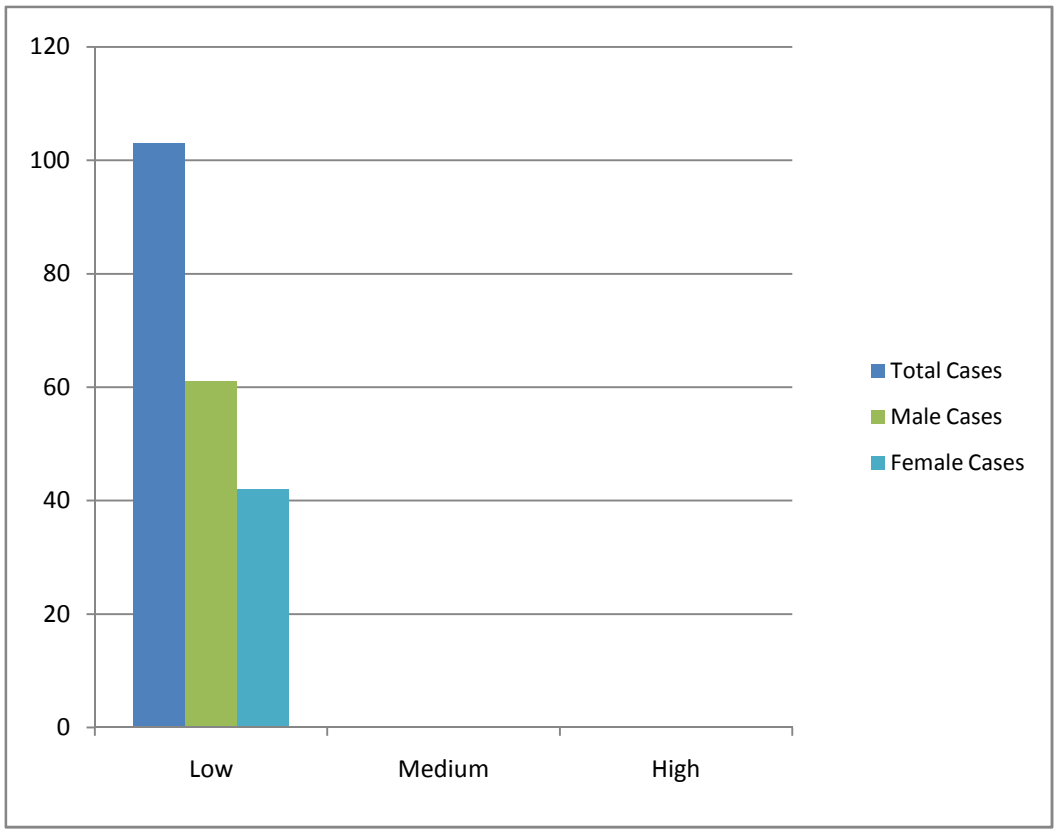
Blood Group	Male		Female	
	Total	%	Total	%
A+ve	22	36.1	17	40.5
B+ve	12	19.7	10	23.8
AB+ve	8	13.1	8	19.0
O+ve	15	24.6	7	16.7
O-ve	2	3.3	0	0.0
AB-ve	1	1.6	0	0.0
A-ve	1	1.6	0	0.0
Total	61	100.0	42	100.0


25
20
15
10
5
0
A+ve
B+ve
AB+ve
O+ve
O-ve
AB-ve
A-ve
Blood Group
Male Total
Female Total

Socioeconomic Status

In the present study all the cases belonged to the low socioeconomic status. The prevalence among high socioeconomic group could not be studied as none of the patients belonged to this strata.

Socio Economic Status	Total		Male		Female	
	Cases	%	Cases	%	Cases	%
Low	103	100	61	59	42	41
Medium	0	0	0	0	0	0
High	0	0	0	0	0	0

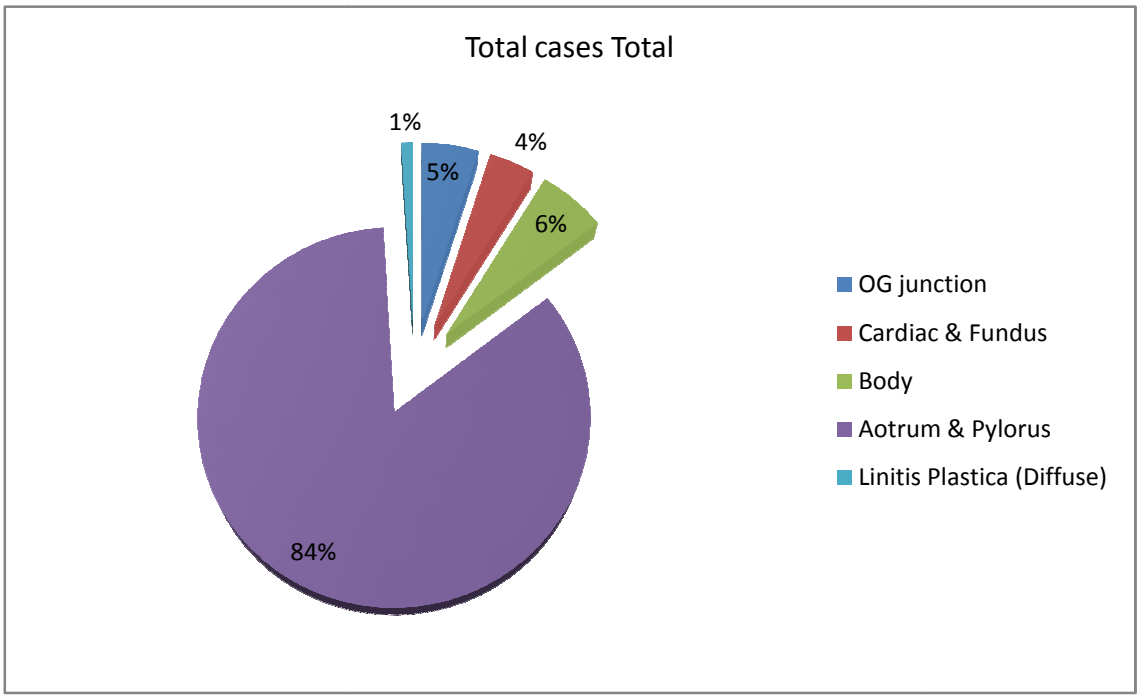

120
100
80
60
40
20
0
Low
Medium
High
Total Cases
Male Cases
Female Cases

Comparison of Site Distribution

In this study the common site of carcinoma stomach was found to be pyloric antrum for both males and females. The total number of males included in this study was 61.Out of this, 55 patients were diagnosed to have pyloric growth. This accounts for 90.2%.Considering the female patients, out of 42 patients, 32 patients were diagnosed as pyloric growth. This accounted for 76.2%.

Sub site	Total cases		Male		Female	
	Total	%	Total	%	Total	%
OG junction	5	4.9	1	1.6	4	9.5
Cardiac & Fundus	4	3.9	1	1.6	3	7.1
Body	6	5.8	3	4.9	3	7.1
Antrum & Pylorus	87	84.5	55	90.2	32	76.2
Linitis Plastica (Diffuse)	1	1.0	1	1.6	0	0.0
Total	103	100.0	61	100.0	42	100.0

Comparison of Site Distribution



Comparison of Environmental Factors

In this study the following environmental factors were analysed, dietary habits, smoking, alcohol, tobacco chewing were taken into account. In dietary habits mixed diet is the main culprit for occurrence of gastric carcinoma. This contributes to 39.9% with relation both genders..In dietary habits spicy food is the major factor which accounts to the occurrence of gastric carcinoma. Next major factor is the consumption of alcohol . In this study 15.2% of male patients had the addiction to alcohol which was the major factor regarding occurrence of gastric carcinoma. Smoking is also the major risk factor accounting to gastric

carcinoma. In this study it has 14.2% of male patients who were taken into count. Tobacco chewing is taken into account in both genders. Contrary to smoking ,in my study female population exceeds male population with regarding to occurrence of stomach carcinoma. Female population accounted for 7.5%,whereas male patients only 3.7% were noted in this study.

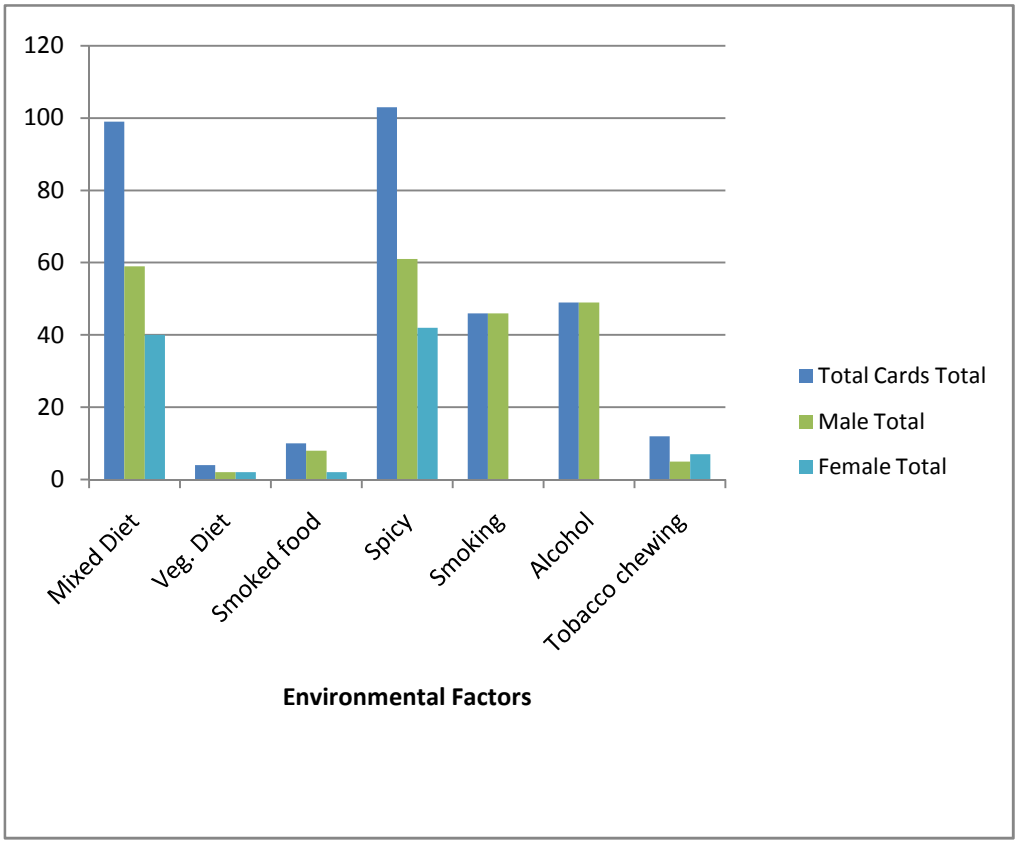


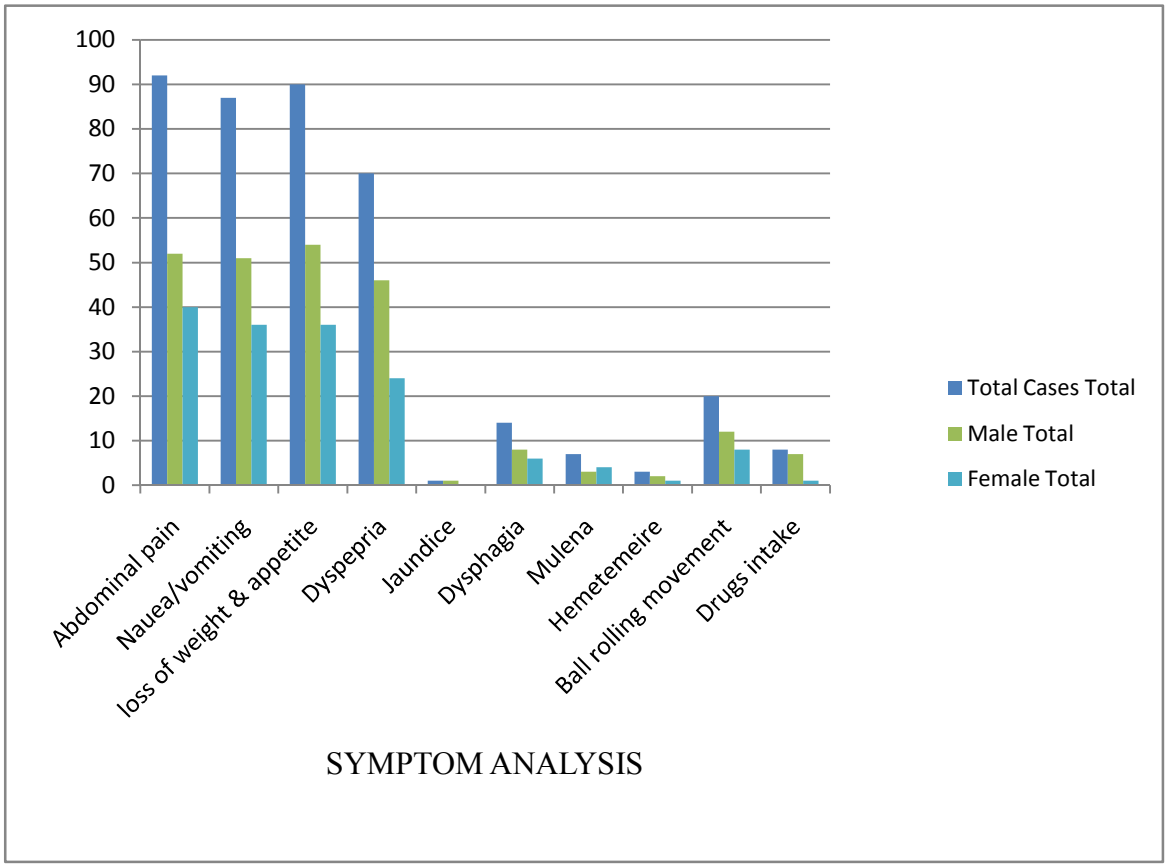
Table showing Comparison of Environmental Factors

Risk Factors	Total cases		Male		Female	
	Total	%	Total	%	Total	%
Mixed Diet	99	30.7	59	25.7	40	43.0
Veg. Diet	4	1.2	2	0.9	2	2.2
Smoked food	10	3.1	8	3.5	2	2.2
Spicy	103	31.9	61	26.5	42	45.2
Smoking	46	14.2	46	20.0	0	0.0
Alcohol	49	15.2	49	21.3	0	0.0
Tobacco chewing	12	3.7	5	2.2	7	7.5
Total	323	100.0	230	100	93	100

Symptom Analysis in the patients of carcinoma stomach

In this study abdominal pain is the most common symptom reported in both genders.This is closely followed by nausea and vomiting, loss of appetite and dyspepsia..Next peculiar symptom is the ball rolling movement, which accounts for 5.1% in both sexes. Dysphagia, malena, hemetemesis and drug intake also contributes to the occurrence of gastric carcinoma.

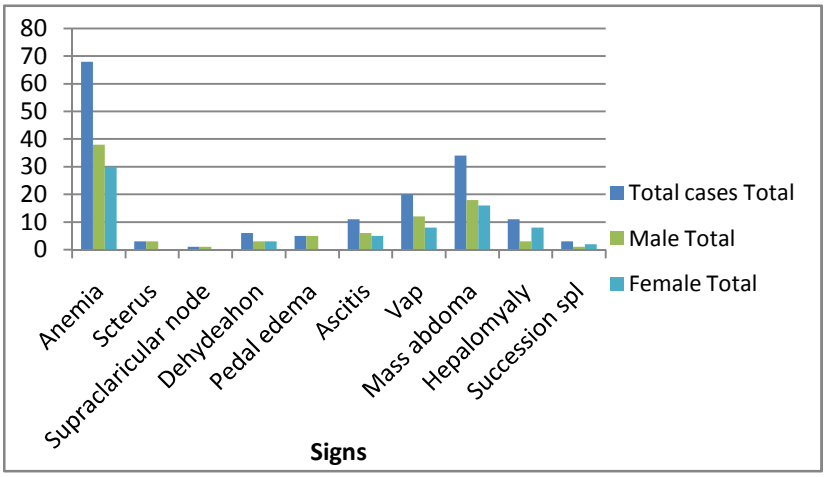
Symptoms	Total Cases		Male		Female	
	Total	%	Total	%	Total	%
Abdominal pain	92	23.5	52	22.0	40	25.6
Nausea/vomiting	87	22.2	51	21.6	36	23.1
loss of weight & appetite		23.0	54	22.9	36	23.1
Dyspepsia	70	17.9	46	19.5	24	15.4
Jaundice	1	0.3	1	0.4	0	0.0
Dysphagia	14	3.6	8	3.4	6	3.8
Malena	7	1.8	3	1.3	4	2.6
Hemetemesis	3	0.8	2	0.8	1	0.6
Ball rolling movement	20	5.1	12	5.1	8	5.1
Drugs intake	8	2.0	7	3.0	1	0.6
Total	392	100.0	236	100.0	156	100.0



Analysis of signs in carcinoma stomach

Among all the cases of carcinoma stomach anaemia was found to be the most common sign, 42.2% in males and 41.7% of females. This was followed byMass abdomen which was reported in 21% of all cases Visible gastric peristalsis is the next common sign, which comes around 12% in both sexes. Ascitis was reported with equal percentage in both sexes. Only one case was reported with presentation of supraclavicular node, that too male patient.

Signs	Total cases		Male		Female	
	Total	%	Total	%	Total	%
Anemia	68	42.0	38	42.2	30	41.7
Icterus	3	1.9	3	3.3	0	0.0
Supraclavicular node	1	0.6	1	1.1	0	0.0
Dehydration	6	3.7	3	3.3	3	4.2
Pedal edema	5	3.1	5	5.6	0	0.0
Ascitis	11	6.8	6	6.7	5	6.9
VGP	20	12.3	12	13.3	8	11.1
Mass abdomen	34	21.0	18	20.0	16	22.2
Hepatomegaly	11	6.8	3	3.3	8	11.1
Succession splash	3	1.9	1	1.1	2	2.8
Total	162	100.0	90	100.0	72	100.0



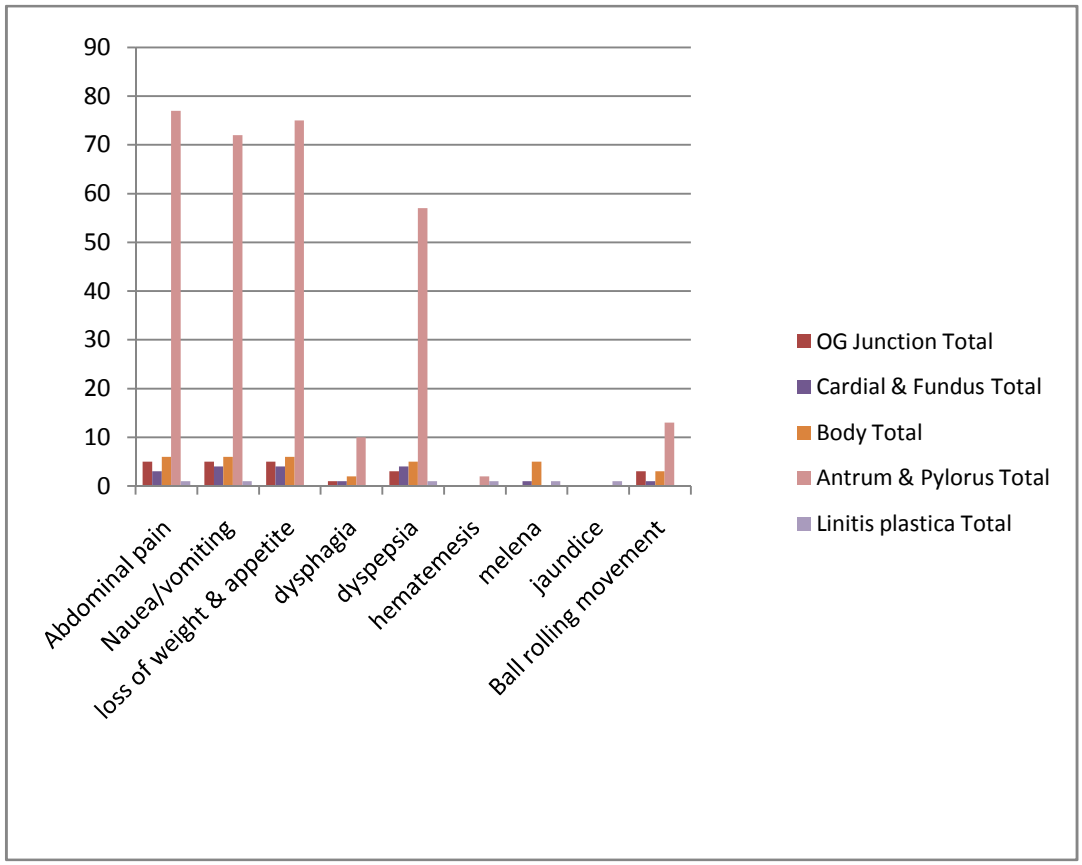
Comparison of Symptoms and site of Tumour

Abdominal pain, nausea, vomiting, loss of weight, dyspepsia and hematemesis were the most common symptoms in patients with lesion in antrum and pylorus.

In patients with OG junction growth, dysphagia was the most common symptom. In cardiac and fundus tumours the common symptoms were nausea, vomiting, loss of weight and dyspepsia. There was one patient with Linitis plastica with symptoms of abdominal pain, nausea, vomiting, hematemesis, jaundice and melena.

Symptoms	Total	OG Junction		Cardial & Fundus		Body		Antrum & Pylorus		Linitis plastic	
		Total	%	Total	%	Total	%	Total	%	Total	%
Abdominal pain	92	5		3		6		77		1	
Nausea / Vomiting	88	5		4		6		72		1	
loss of weight & appetite	90	5		4		6		75		0	
Dysphagia	14	1		1		2		10		0	
Dyspepsia	70	3		4		5		57		1	
Hematemesis	3	0		0		0		2		1	
Malena	7	0		0		1		5		1	
Jaundice	1	0		0		0		0		1	
Ball rolling movement	20	3		1		3		13		0	
Total		22	0	17	0	28	0	304	0	6	0

Comparison of Symptoms and site of Tumour

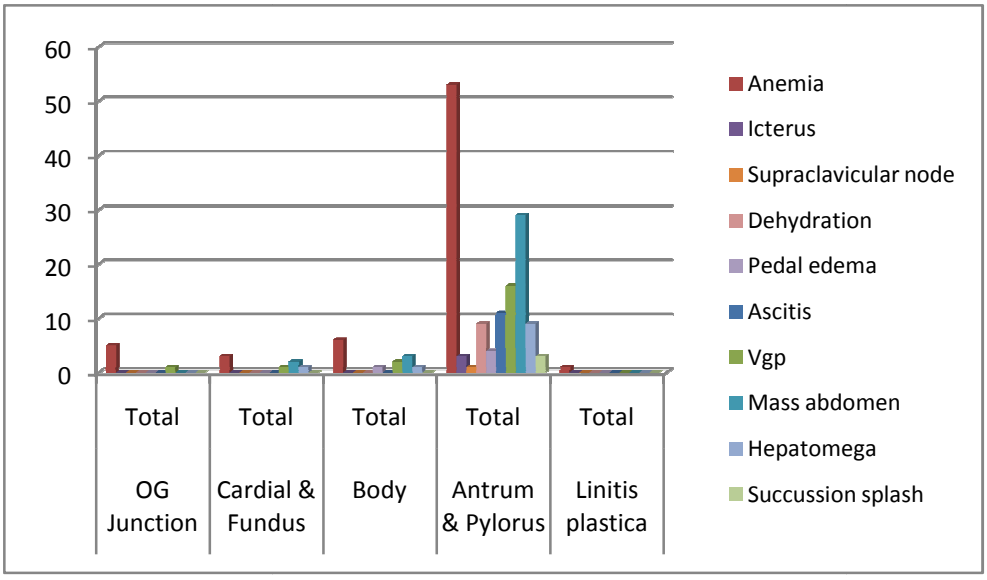


Analysis of site of tumor with signs

Anaemia was the most common sign in cases with tumour in any part of the stomach. Followed by mass abdomen, visible gastric peristalsis and ascitis which were most common in cases with tumor in antrum and pylorus. Supraclavicular node was present in one case of tumor in antrum.

Signs	OG Junction		Cardial & Fundus		Body		Antrum & Pylorus		Linitis plastica		Total
	Total	%	Total	%	Total	%	Total	%	Total	%	
Anemia	5	3.0	3	1.8	6	3.6	53	32.1	1	0.6	68
Icterus	0	0.0	0	0.0	0	0.0	3	1.8	0	0.0	3
Supraclavicular node	0	0.0	0	0.0	0	0.0	1	0.6	0	0.0	1
Dehydration	0	0.0	0	0.0	0	0.0	9	5.5	0	0.0	9
Pedal edema	0	0.0	0	0.0	1	0.6	4	2.4	0	0.0	5
Ascitis	0	0.0	0	0.0	0	0.0	11	6.7	0	0.0	11
VGP	1	0.6	1	0.6	2	1.2	16	9.7	0	0.0	20
Mass abdomen	0	0.0	2	1.2	3	1.8	29	17.6	0	0.0	34
Hepatomegaly	0	0.0	1	0.6	1	0.6	9	5.5	0	0.0	11
Succussion splash	0	0.0	0	0.0	0	0.0	3	1.8	0	0.0	3
Total	6	3.6	7	4.2	13	7.9	138	83.6	1	0.6	

Representation of Signs with Relation Side

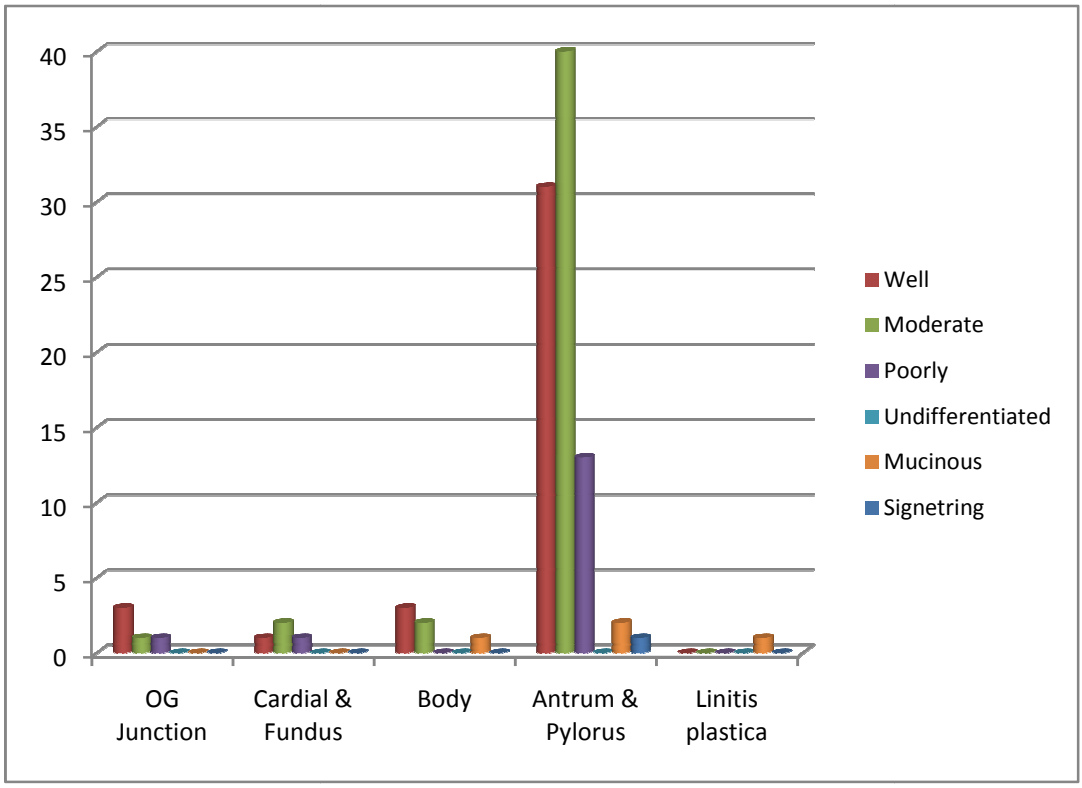


Comparison of Histology

Histopathological analysis was done for all resected tumors. In this study the most common type was found to be moderately differenciated tumour. This accounts to 41% in males and 47.6% in females. Next common type reported was well differentiated type, which accounted to 44.3% in males, and 26.2% in females. Female patients has maximum number of poorly differentiated type. The least common types reported was undiffrentiated, mucinous and signet ring types .

Histology	Study		Male		Female	
	Total Cases	%	Cases	%	Cases	%
Well	38	36.9	27	44.3	11	26.2
Moderate	45	43.7	25	41.0	20	47.6
Poorly	15	14.6	5	8.2	10	23.8
Undifferentiated	0	0.0	0	0.0	0	0.0
Mucinous	4	3.9	3	4.9	1	2.4
Signet ring	1	1.0	1	1.6	0	0.0
Total	103	100.0	61	100.0	42	100.0

Histological Representation



Comparison of histology & the site of the tumour

Most of the OG junction tumors were found to be well differentiated. Tumors in Cardia, fundus, body, antrum and pylorus were mostly moderately differentiated tumors.

The case with linitis plastic was found to be mucinous tumor in histopathology. One case of signet ring tumor was seen in a case of tumor at the pylorus and antrum.

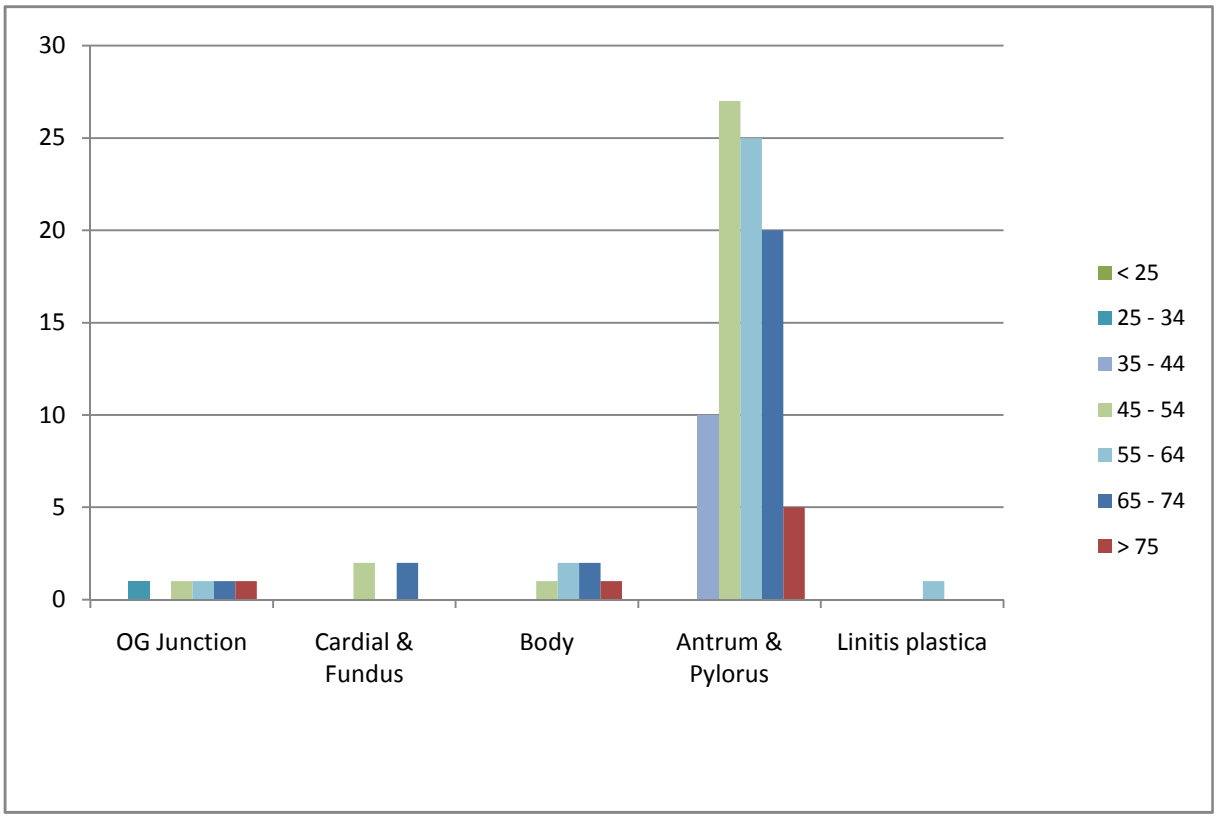
Histology	OG Junction	Cardial & Fundus	Body	Antrum & Pylorus	Linitis plastica	Total	%
Well	3	1	3	31	0	38	36.9
Moderate	1	2	2	40	0	45	43.7
Poorly	1	1	0	13	0	15	14.6
Undifferentiated	0	0	0	0	0	0	0.0
Mucinous	0	0	1	2	1	4	3.9
Signet ring	0	0	0	1	0	1	1.0
Total	5	4	6	87	1	103	100.0

Comparison of age group & site of tumour

Tumors in the OG junction were most common in the age group of above 45 years of age. Tumors in the cardia and fundus were common in age group of 45-54 years and 65-74 years. Tumors in the body of stomach were common in cases above the age of 55 years. Tumors in the antrum and pylorus were found most commonly in the age group of 45-54 years.

Age Group	OG Junction		Cardia & Fundus		Body		Antrum & Pylorus		Linitis plastica	
	Total	%	Total	%	Total	%	Total	%	Total	%
< 25	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
25 – 34	1	20	0	0.0	0	0.0	0	0.0	0	0.0
35 – 44	0	0.0	0	0.0	0	0.0	10	11.5	0	0.0
45 – 54	1	20	2	50	1	16.7	27	31.0	0	0.0
55 – 64	1	20	0	0.0	2	33.3	25	28.7	1	100
65 – 74	1	20	2	50	2	33.3	20	23	0	0.0
> 75	1	20	0	0.0	1	16.7	5	5.7	0	0.0
Total	5	100	4	100	6	100	87	100	1	100

Age group and site of Tumor



DISSCUSSION

Gastric carcinoma remains to be one of the important malignant diseases, with significant geographical, ethnic and socioeconomic differences in distribution. Once it was considered as the second most common malignancy in the world, now it is on decreasing trend. This may be due to well proven facts like recognition of H.pylori, improved dietary habits, low intake of preserved foods, salt etc. In western countries upper gastric carcinoma predominates, whereas in India antral growth was found to be the most common presentation.

Although worldwide the incidence is on decreasing trend, in India Gastric carcinoma, still a major malignant disease which has an impact over low socioeconomic population. So with relevance to these facts this study was undertaken to analyse the various factors influencing the incidence of Gastric carcinoma and various treatment modalities available in Department of General Surgery, Tirunelveli Medical College Hospital.

(i) Sex distribution:

(ii) Gastric carcinoma is more common in males with global age standardised incidence for males about 2.2 times higher than females. In my study group 59% of cases were males and 41% were females, compared to result of sumathi et al 2009.

(iii) Age distribution: Age wise trends in carcinoma stomach have been reported worldwide being largely a disease of older age group in most countries. In this study maximum number of cases were seen above age of 45 years. This finding correlates with the study done Urmi Sen et al in eastern India, which also reported a similar age trend with increasing incidence of Gastric carcinoma with age.

In this study the youngest was aged 25 and oldest was 76 years of age.

(iv) Socioeconomic status: As the study was conducted in a government hospital, most of the cases belonged to low socioeconomic status. The scenario is similar across india where majority of population belonging to the lower socioeconomic status. The results were comparable to results of gajalakshmi et al1995.

(v) Blood group: The association of blood group A is well known and the same results were obtained in this study. Other blood groups B positive and O positive was noted to be on lower side on occurrence of gastric

carcinoma. In this study Rh negative group patients were found to be on least occurrence. The results were comparable with studies by kamalesh guleria et al, Punjab and jose et al,kerala.

(vi) Environmental factors: Gastric cancer is known to be associated with several environmental factors among which diet has an important role. The association of diet has been studied extensively by various authors. The results of this study were similar to those obtained by sumathi et al, in which spicy diet and mixed food were found to be major environmental risk factors Alcohol consumption and smoking among the male patients was the next major factor which contributes to the increasing incidence of Gastric carcinoma. Tobacco chewing also found to be one the environmental factors which has an impact on the rising trend of Gastric carcinoma, especially among female patients. All these findings in this study were well comparable with study done by Sumathi et al.

(vii) Symptoms : Abdominal pain was the most common symptom reported in ninety two of the cases compared to 56.6% in the study done by Safee et al. Next common symptoms noted were nausea and vomiting, loss of appetite/weight and dyspepsia. These findings also well

correlates along with the study done by Safee et al. Dysphagia, malena, hemetemesis are the symptoms with least occurrence in this study.

(viii) Signs : In this study anemia was the most common sign noted in both sexes. Although anemia is the common finding among the population here,this finding correlates similar to study done by Kaiser Jamil et al.Next common signs were mass abdomen and visible gastric peristalsis.Ascites was also noted in few cases.

(ix) Site specific presentation: In this study, Pylorus and antrum were the most common site of occurrence of gastric cancer. This accounts to 81% of total cases, and this findings are well comparable with study done by Cherian et al.

(x) Histology : In this study most of the cases were moderately differentiated as compared to safe et al which constituted poorly differentiated tumors

SUMMARY

Gastric carcinoma is the disease of old age as the incidence increases with advancing age. The incidence has declined in the world because of changing diet, food preparation and environmental factors. This study was done to study the incidence of gastric carcinoma in Tirunalveli medical college hospital Tirunelveli.

The incidence of gastric carcinoma is more among males(3:2). The age of incidence ranges from 25 – 75 years, most commonly over 45 years. The youngest being at the age of 25 years. Increased incidence of carcinoma stomach was found in patients of both sex taking spicy food. Smoking and Alchohol also contributes the occurrence of Gastric carcinoma.

The patients with A positive blood group were found to be more susceptible to gastric carcinoma. The common symptoms presented in this study were abdominal pain, nausea and vomiting, loss of appetite/weight. Anemia, mass abdomen and visible gastric peristalsis were the common signs. This shows the patients with advanced stage are turning up to the hospital.

The most common site observed were the pylorus and antrum. The male patients commonly showed well differentiated tumors, whereas the

females had higher incidence of moderately differentiated tumors. Most of the well differentiated tumors were found in the oesophagealgastric junction, whereas pyloric antal tumors showed moderate differentiation.

As most of the patients in the study presented in advanced stages treatment was palliative in the form of gastro jejunostomy and jejunojejunostomy.

CONCLUSION

Gastric cancer mortality rate have remained relatively unchanged over the years in India, inspite of various investigative and treatment modalities. This may be due late detection of disease, which is the important factor in relation to the prognosis and outcome of the disease. Although there are lot of screening investigations for early detection of gastric carcinoma, in India due to cost benefit ratio it becomes impossible .This was well noted in this study as the most of the patients were presented with advanced symptoms and signs. So the palliative treatment was the major treatment modality observed in this study. So in future ,early detection of of Gastric carcinoma should be our main objective, so that we can reduce the mortality rate, and importantly to improve the quality of life in patients suffering from Gastric carcinoma

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PROFORMA
Name:
Age:
Sex:
IP no:
Occupation:
Socioeconomic status:
Address:
Date of Admission:
Date of surgery :
Date of Discharge:
Complaints:
Mass Abdomen:
- Duration;
- Size:
- Site;

Pain abdomen:
- Site ;
- Nature:
- Duration:
- Radiating nature:
- Aggravating factors:
- Relieving Factors:

Vomitting:
- Nature:
- Spontaneous/ Induced;
- Colour:
- Content:
- Foul smelling:
- Blood Stained:

Dysphagia:
- Duration:
- Solids/ Liquids / Both

Dyspepsia:
H/o Ball rolling movements
H/o Loss of weight/Loss of apetite
H/o Weakness
H/o Abdominal distension
H/o Jaundice
H/o Hemetemesis / Melena

H/o Fever
H/o Pruritus
H/o Drug intake
Past History:
DM /HT / TB / Bronchial Asthma / IHD
H/o gastritis & treatment
H/o previous surgery
H/o Blood transfusion
Family History:
Personal History:
Dietary Habits : Veg / Non veg
- High consumption of animal fat
- High salted & pickled food
- Smoked food

H/o smoking – No.of cigarettes / day & duration
H/o alchohol intake-Quantity & duration
H/o Tobacco chewing
Bowel & Bladder habits
Menstrual History :
General Examination:
- Nutrition
- Hydration status
- Anemia
- Jaundice
- Pedal edema
- Lymph nodes

Per Abdomen Examination:
Inspection-Site of mass
- Size
- Shape
- Movement with respiration
- Umbilicus
- VGP
- VIP
- Venous pulsation
- Ascites
- Scar

Palpation – Consistency

Warmth

Tenderness

Confirmation of Inspection Findings

Guarding & Rigidity

Organomegaly

Percussion :

Liver Dullness

Over the mass

Shifting Dullness

Fluid thrill

Free Fluid

Auscultation: Bowel sounds

Succession splash

Bruit

Digital rectal examination:

Other systems :

CVS, RS , CNS, LOCOMOTOR

CLINICAL DIAGNOSIS:

Investigations:

Urine Albumin, sugar, deposits

Complete Hemogram

Blood Gr & Typing

Blood Sugar, urea, creatinine, electrolytes

LFT

CXR

X-ray Abdomen

ECG

FNAC

UGI Endoscopy & Biopsy

USG Abdomen & Pelvis

CT Abdomen

HPE

Barium meal in selective cases

Pre operative treatment Given – RT, CT

Operative Notes:

Post Operative Follow up.

Day of Ryle's tube removal

Day of suture removal

Complications

Chemotherapy

SNO	NAME	RELIGION	IP NO	AGESEX	ECO STATUS	smoking	alchohol	DIET	SITE	PAIN	VOM	Dysphagia	Ballrolling	loss of wt	Dyspepsia	Anemia	icterus	Hydration	ped edem	Sc node	mass	VGP	Ascites	liver engl	Blood gp	USG
1	Gobala krishnan	H	40525	58M	L	+	+	M	A	+	-	-	+	-	+	-	-	+	-	-	+	-	-	-	A+	AW thickening
2	Leelavathi	H	13322	50F	L	-	-	V	CF	+	+	-	-	+	-	+	-	-	-	-	+	+	-	-	B+	Growth B and F
3	Muthiah	H	3663	70M	L	+	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	+	+	-	O+	Growth body
4	Jeyaraj	H	2845	60M	L	+	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	O+	AW thickening
5	Santhanam	H	47408	60F	L	-	-	M	OG	+	+	+	+	+	-	+	-	-	-	-	-	-	+	-	A+	Growth OGJ
6	Francis	C	29859	55M	L	+	+	M	A	+	+	+	+	+	+	+	-	-	+	-	-	-	-	-	AB+	Dilated stomach
7	Poolachi	H	21595	40F	L	-	-	V	A	+	-	-	+	+	-	+	-	+	-	-	+	-	-	-	A+	AW thickening; PP nodes +
8	Pandi	H	33651	57M	L	+	+	M	A	+	-	-	-	+	+	+	-	-	-	-	-	+	-	-	A+	AW thickening
9	Kalimuthu	H	55467	58M	L	+	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	O+	AW thickening
10	Ponaiya	H	61855	60M	L	+	+	M	PA	+	+	-	-	-	-	+	-	-	-	-	+	-	-	-	A+	PA growth
11	Susila	H	12822	50F	L	-	-	M	OG	-	+	+	-	-	-	+	-	-	-	-	+	+	-	-	A+	OGJ growth
12	Muthiah	H	37677	57M	L	+	-	V	P	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	AB+	Pylorus growth

13	Kumar	H	15487	47M	L	+	+	M	P	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	-	B+	Ulcerative growth pylorus
14	Chellapandi	H	3668	40M	L	+	+	M	P	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	O+	Ulcerative pylorus growth
15	Periakannan	H	61852	60M	L	+	+	M	P	+	+	-	+	+	+	+	-	-	-	-	+	+	-	-	AB+	PA growth
16	Subbaiah	H	59597	65M	L	+	+	M	A	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	O+	AW thickening with GOO
17	Ponnusamy	H	53848	55M	L	+	+	M	P	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	B+	Ulcerative growth pylorus
18	Murugesan	H	56309	49M	L	+	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	O+	Distended stomach with GOO
19	Sundar	H	39244	48M	L	+	+	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	B+	Growth antrum GOO
20	Nagaraj	H	13810	45M	L	+	+	M	A	+	+	+	-	+	+	+	-	-	+	-	+	+	+	-	A+	Distended stomach with GOO
21	Gopal kambar	H	40525	58M	L	-	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	A+	Growth antrum GOO
22	Paulraj	C	10143	72M	L	+	+	M	A	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	A+	PA growth extending to proximal part
23	Amirthapuspham	H	31318	58F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	+	+	-	+	A+	AW thickening with GOO
24	Chandra	H	11224	40F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	B+	Antral growth
25	Ramalakshmi	H	48512	53F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	A+	Growth antrum GOO
26	Fathima	M	29246	54F	L	-	-	M	A	+	+	-	-	-	-	+	-	-	-	-	+	-	+	+	B+	Antral growth;PPNLA
27	Pappa	H	47314	46F	L	-	-	M	B	+	+	-	+	+	-	+	-	-	-	-	-	-	-	+	B+	UP growth body
28	Valli	H	34517	65F	L	-	-	M	A	+	+	+	-	+	+	+	-	-	-	-	-	+	-	-	A+	AW thickening with GOO

29	Shanmugavadivu	H	31722	65F	L	-	-	M	A	+	+	-	+	+	+	+	-	-	-	-	-	-	-	-	B+	AW thickening with GOO
30	Gracie	C	38296	70F	L	-	-	M	A	+	-	-	-	+	+	+	-	-	-	-	+	-	-	-	AB+	AW thickening with GOO
31	Thangamani	H	56449	70F	L	-	-	M	A	+	+	-	-	+	-	+	-	-	-	-	+	-	-	-	O+	Growth pylorus
32	Chelliah	H	40952	65M	L	-	+	M	A	+	+	-	+	+	+	-	-	-	-	-	-	+	+	-	B+	Gastric wall thickening;PF;LS;ascitis
33	Elias	C	42512	55M	L	-	+	M	A	+	+	-	+	+	-	-	+	-	-	-	-	-	-	-	O+	Growth antrum
34	Subramaniyan	H	48622	59M	L	+	+	M	A	-	+	-	-	+	+	+	-	-	-	-	-	-	+	-	A+	Growth antrum
35	Chellappa	H	27574	58M	L	-	+	M	A	+	+	-	-	+	+	-	-	-	-	-	-	+	-	-	B+	Growth antrum
36	Ramasamy	H	30969	62M	L	-	+	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	AB-	AW thickening;minimal ascitis
37	Mariappanadar	H	6963	80M	L	+	+	M	A	+	+	-	-	-	+	+	-	-	-	-	-	+	-	-	B+	AW thickening
38	Kanagarani	H	5081	45F	L	-	-	M	A	+	+	-	+	+	-	+	-	-	-	-	+	-	-	-	AB+	AW thickening
39	Chendu	H	40905	45F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	+	+	-	-	AB+	AW thickening
40	Ramalakshmi	H	7805	49F	L	-	-	M	A	+	+	+	-	+	+	+	-	-	-	-	+	-	-	-	O+	NS
41	Kamalam	H	15031	49F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	+	+	A+	AW thickening
42	Esakkiammal	H	58250	44F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	+	-	-	AB+	Growth antrum;ascitis
43	Saraswathy	H	47135	47F	L	-	-	M	A	+	+	-	-	-	-	+	-	-	-	-	+	-	-	-	A+	AW thickening
44	Arunachalam	H	32247	65M	L	-	+	M	OG	-	+	-	-	+	+	+	-	-	-	-	-	-	+	-	A+	Growth PA
45	Ramasamy	H	57322	47M	L	+	+	M	B	+	+	-	-	+	+	+	-	-	-	-	+	-	-	+	B+	Growth body;LS

46	Muthiah	H	36663	70M	L	+	+	M	B	+	-	-	+	+	+	+	-	-	+	-	-	-	-	-	AB+	Growth body
47	Chellaiah	H	49419	70M	L	+	+	M	B	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	AB+	? CA stomach
48	Mariappan	H	52278	45M	L	+	+	M	A	+	-	+	-	+	+	+	+	-	-	-	+	-	-	-	A+	AW thickening;PPNLA
49	Ramasamy	H	48509	65M	L	+	+	M	A	+	+	-	-	+	+	+	-	-	-	-	-	+	-	-	A+	Diffuse circumferential AW thickening
50	Arunachalavadivu	H	8346	66F	L	-	-	M	CF	+	-	-	+	+	-	+	-	+	-	-	+	+	-	-	A+	UP growth B and F
51	Shanmugavel	H	19947	62M	L	+	+	M	A	+	+	-	+	+	+	+	+	-	-	-	-	-	-	-	O+	? CA stomach
52	Moorthy	H	35235	48M	L	+	+	M	A	-	+	-	-	+	+	+	-	-	-	-	-	-	-	-	B+	Antral growth
53	Ulaganathan	H	5341	65M	L	+	+	M	A	+	+	-	-	+	+	+	-	-	-	-	+	-	-	-	B+	Gastric wall thickening at pyloric antrum
54	Arumugathai	H	3104	25F	L	-	-	M	OG	+	+	+	-	+	+	+	-	-	-	-	+	-	-	+	A+	Hepatomegaly with PPNLA
55	Mailerumperumal	H	59459	59M	L	-	-	M	A	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	O+	Gastric wall thickening at pyloric antrum
56	Pandian	H	58068	65M	L	+	+	M	A	+	+	-	+	+	+	+	-	-	+	-	+	-	-	-	B+	AW thickening
57	Sundaram	H	57450	65M	L	+	+	M	A	+	+	-	-	+	-	+	-	-	-	-	-	-	-	-	A+	AW thickening
58	Rathinasamy	H	26665	35M	L	+	+	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	AB+	Dilated stomach with GOO
59	Krishnan	H	9304	80M	L	+	+	M	A	+	+	-	-	-	+	+	-	-	-	-	-	-	-	+	A+	Hypoechoeic mass epigastric region
60	Subramaniyan	H	43728	58M	L	+	+	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	A+	Pyloric growth
61	Sokkalingam	H	27941	65M	L	+	+	M	A	+	+	-	-	+	+	-	-	-	-	-	+	+	-	-	A+	Dilated stomach

62	Philip	C	23954	60M	L	+	+	M	A	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	AB+	AW thickening
63	Guruvaiah	H	49002	60M	L	+	+	M	A	+	+	-	-	+	+	+	-	+	-	-	+	-	-	-	O+	Distended stomach with GOO
64	Subramaniyan	H	18582	72M	—–L	+	+	M	A	+	+	+	-	+	+	-	-	-	-	-	+	-	-	-	O+	AW thickening
65	Vimalalincythilagam	C	33249	61F	L	-	-	M	B	+	+	-	-	+	-	+	-	-	-	-	+	-	-	-	A+	UP growth body
66	Kallapiran	H	22920	42M	L	+	-	M	A	-	+	-	-	+	+	-	-	-	-	-	+	-	-	-	O+	AW thickening
67	Vasudevan	H	25385	49M	L	+	-	M	A	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	B+	AW thickening
68	Rama lakshmi	H	54213	53F	L	-	-	M	B	+	+	-	+	-	-	+	-	-	-	-	-	-	-	+	B+	Ulcerative growth antrum
69	Samuvelraj	C	57493	70M	L	+	+	M	A	+	+	-	+	+	+	-	-	-	-	-	-	-	-	-	A+	AW thickening;distented stomach
70	Madathy	H	32353	45F	L	-	-	M	A	+	+	-	-	+	-	+	-	-	-	-	+	-	-	-	B+	AW thickening
71	Kajamydeen	M	32246	65M	L	+	+	M	A	-	+	-	+	-	-	+	-	-	-	-	-	-	-	-	A+	PA growth
72	Shanmugathai	H	45663	54F	L	-	-	M	A	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	A+	Pyloric wall thickening
73	Kallimuthu	H	53676	48M	L	+	-	V	A	-	+	-	-	+	-	-	-	-	+	-	+	-	-	-	A+	Pyloric wall thickening
74	Palani	H	2033	48M	L	+	+	M	A	+	-	-	-	+	+	+	-	-	-	-	-	-	-	-	AB+	Dilated stomach with LS
75	Velsamy	H	42487	77M	L	+	+	M	A	+	+	-	-	+	+	-	-	-	-	-	+	-	-	-	A+	AW thickening with LS
76	Velayutham	H	55884	51M	L	+	+	M	A	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	B+	AW thickening;PPNLA
77	Karupaiyee	H	57190	65F	L	-	-	M	CF	+	+	-	-	+	+	-	-	-	-	-	-	-	+	+	B+	Growth cardia

78	Selvasekaran	H	1389	50M	L	+	+	M	A	+	-	+	-	+	+	-	-	-	-	-	-	-	-	-	O+	AW thickening;ascitis
79	Selvi	H	13947	37F	L	-	-	M	A	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	O+	Antrum thickening
80	Rabiya	M	48519	65F	L	-	-	M	A	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	AB+	PAW thickening;LS
81	Aadhinachiar	H	16649	55F	L	-	-	M	A	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	AB+	Pylorus growth
82	Poornam	H	39569	40F	L	-	-	M	A	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	B+	AW thickening
83	Muthulakshmi	H	31290	47F	L	-	-	M	A	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	A+	Growth pyloric antrum
84	Karupasamy	H	56850	75M	L	-	-	M	F	+	-	-	-	+	+	+	-	+	-	-	-	-	-	-	O+	Growth fundus extending upto OGJ with ascitis
85	Kanagaraj	H	45537	58M	L	+	+	M	P	+	+	-	+	+	-	+	-	-	-	-	-	-	-	-	A+	NS
86	Chellammal	H	14048	44F	L	-	-	M	A	+	-	-	-	+	+	+	-	-	-	-	-	-	-	-	B+	LS; AW thickening
87	Saraswathi	H	17481	45F	L	-	-	M	A	+	+	-	+	+	+	+	-	-	-	-	-	+	-	-	AB+	Growth antrum with GOO
88	Subbiah	H	8092	43M	L	-	-	M	P	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-	A-	Growth pylorus
89	Rajammal	H	4398	60F	L	-	-	M	P	+	+	-	-	+	+	-	-	-	-	-	-	-	-	-	AB+	AW thickening
90	Petchiammal	H	57476	47F	L	-	-	M	A	+	+	-	-	-	+	-	-	-	-	-	-	-	+	+	A+	LS
91	Neela	H	25442	60F	L	-	-	M	A	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	O+	AW thickening
92	Annababu	H	50670	50F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	A+	Growth antrum

93	Sudalaimadi	H	3758	45F	L	-	-	M	A	+	+	-	-	+	+	-	-	-	-	-	-	+	-	-	O+	Pyloric growth
94	Angammal	H	38554	41F	L	-	-	M	P	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	O+	AW thickening
95	Guruvammal	H	56294	75F	L	-	-	M	A	+	+	-	-	+	+	+	-	-	-	-	-	-	-	-	A+	Growth antrum
96	Chellammal	H	50038	55F	L	-	-	M	OG	-	+	-	-	+	+	+	-	-	-	-	+	+	-	-	A+	Growth C and F extending to OGJ
97	Soma sundaram	H	43282	61M	L	+	+	M	P	+	-	-	+	+	+	-	-	-	-	-	-	-	-	-	A+	Pyloric growth
98	Balammal	H	54680	52F	L	-	-	M	A	+	+	-	+	+	+	-	-	+	-	-	-	-	-	-	O+	Ascitis
99	Thavasirajan	H	35103	60M	L	-	-	M	A	+	-	-	-	+	+	+	-	-	-	-	+	-	-	-	O+	AW thickening
100	Esakki	H	36288	40M	L	-	-	M	A	+	+	+	-	+	-	-	-	-	-	-	-	-	-	+	A+	AW growth
101	Kaveri	H	48535	55M	L	-	-	M	P	+	+	-	-	-	+	+	-	-	-	+	+	-	-	-	A+	Antral growth
102	Ramaiah	H	5863	70M	L	-	-	M	P	+	+	+	-	+	+	-	-	-	-	-	+	-	-	-	O-	Growth antrum
103	Palpandi	H	10046	85M	L	-	-	M	P	+	+	+	-	+	-	+	-	-	-	-	+	+	+	-	O-	Growth antrum